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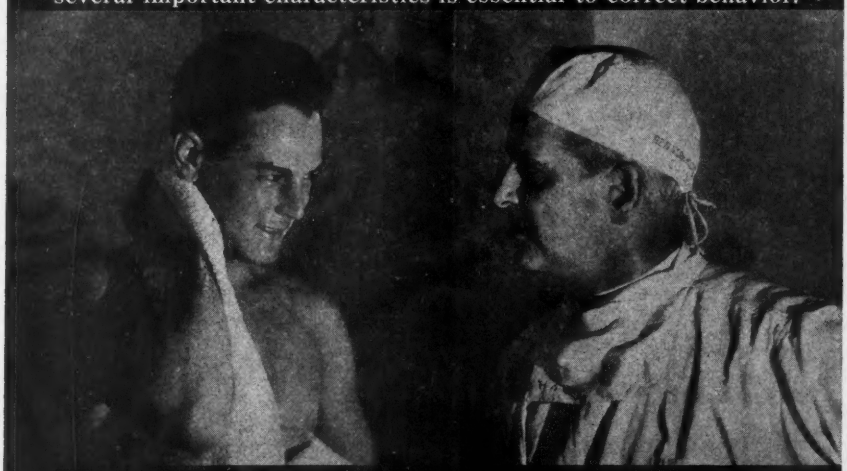
"A suture can't be judged by any one feature—a combination of several important characteristics is essential to correct behavior."



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The Journal of Thoracic Surgery

VOL. 4 AUGUST, 1935 NO. 6

Original Communications

PUTRID ABSCESS OF THE LUNG FOLLOWING DENTAL OPERATIONS*

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NEW YORK, N. Y.

INTRODUCTION

PULMONARY abscess following dental operations is regarded as a relatively rare complication. Its incidence is generally pictured as a sequel to aspiration of a tooth following extraction. Fifty-one cases are mentioned in recent American literature.[1-12] In practically all of the reports aspiration of tooth roots or whole teeth during general anesthesia was the assumed etiologic factor.

Neither the incidence of pulmonary abscess following extraction under general anesthesia without evidence of tooth aspiration nor of its occurrence following dental operations under local anesthesia has been adequately analyzed. That such a sequence may be not infrequent is indicated by a series of twelve cases of abscess of the lung occurring shortly after dental procedures in which there was no clinical or roentgenologic evidence of tooth aspiration. These cases are the basis of the present report. Eight of them followed extraction of teeth under local anesthesia.

The seriousness of abscess of the lung warrants a critical study of this hitherto unemphasized etiologic factor, and to this end an attempt has been made to analyze the mechanism involved and to suggest some preventive measures. The important point to be made is that pulmonary abscess may and does occur after tooth extraction without aspiration of the tooth itself and that it is directly related to the dental operation.

*From the Surgical Service of Dr. Harold Neuhof and the Medical Services, Mount Sinai Hospital, New York City.
Received for publication June 11, 1935.

ETIOLOGIC GROUPING

The more recent literature indicates that about 25 per cent of pulmonary abscesses follow surgical operations, chiefly in the mouth and throat.[4, 6, 8, 10-18] A small percentage is reported as following comatose states caused by drugs or disease. The largest group is generally classified as of unknown etiology. Table I represents a classification of seventy consecutive cases of abscesses of the lung studied in Mount Sinai Hospital.

TABLE I

ETIOLOGY	NUMBER OF CASES	PERCENTAGE
Posttonsillectomy	11	16
Postoperative (other)	3	4
Postsubmersion	1	1
Unknown	55	79

It should be noted, however, that a correct allocation of the cases with the etiology ascribable to dental procedures to be described raises sharply the percentage of pulmonary abscesses following surgical operations (Table II). It can be assumed, therefore, that a comparable reduction in the statistical representation of the group of unknown etiology in other reports might have resulted if the possibility of an etiology consequent to dental operations had been carefully considered.

TABLE II

ETIOLOGY	NUMBER OF CASES	PERCENTAGE
Posttonsillectomy	11	16
Postoperative (dental)	12	17
Postoperative (other types)	3	4
Postsubmersion	1	1
Unknown	43	61

MECHANICS GOVERNING FORMATION OF ABSCESS OF THE LUNG

In postoperative pulmonary abscess two routes through which infection may reach the lungs are recognized: (1) the transmission of embolic material from a focus via the blood stream and (2) direct aspiration of infected particulate material. The latter hypothesis finds such abundant support in so many clinical observations and necropsy findings that it is accepted by the group studying thoracic diseases at Mount Sinai Hospital as the usual mechanism.

Some of the factors establishing this assumption are: (a) the ease with which particulate matter in the mouth may be aspirated, (b) the similarity of the common bacteriologic findings (particularly anerobes) in abscess of the lung with the flora associated with the so-called pyorrhea pocket, (c) the occurrence of a large single (occasionally lobulated) abscess as differentiated from the small multiple abscesses of pyemia, (d) the general cortical location of abscesses and their open communication

with smaller bronchi, (e) the rarity with which infarcts are found, and (f) the rarity of anaerobic septicemia.

The occurrence of aspiration from the oral cavity under varying conditions is shown in a number of clinical and experimental reports. Mullins and Ryder[19] demonstrated the readiness with which carbon particles are aspirated from the mouth in normal unanesthetized rabbits. Immediate bronchoscopy by Myerson[20] after tonsillectomy under general anesthesia in a series of children disclosed blood below the vocal cords in 76 per cent of the cases. According to Nather,[21] whose work was corroborated by Iglauer,[22] and by Ochsner and Nesbit,[23] patients with cocainized pharynges invariably aspirate a large proportion of iodized oil when they attempt to swallow the substance. It is a matter of routine observation at Mount Sinai Hospital to note that the swallowing of iodized oil without preliminary pharyngeal anesthesia often permits aspiration of a portion of the fluid. Iglauer[24] also reported bronchoscopic findings of blood in the larynx and trachea of 20 per cent of a series of 50 adults directly after tonsillectomy under local anesthesia.

The similarity of the anaerobic organisms recovered in a large percentage of abscesses of the lung with those associated with pyorrhea is emphasized by Smith,[25] Bucher,[16] and many others. Crowe and Searf,[26] as well as Smith,[27] describe the experimental production of pulmonary abscess in laboratory animals by the introduction into the trachea and bronchi of material saturated with scrapings obtained from human pyorrhea pockets. Cohen[28] has been successful in such experiments on dogs when a certain amount of blood was introduced with the organisms.

ASPIRATION DURING DENTAL OPERATIONS

The usual mechanism resulting in pulmonary abscess, as explained by Wessler,[14] is that aspiration of particulate infected matter occurs during the course of operations (or in states of unconsciousness however induced). Coagulation of blood, if blood is simultaneously present, seals the small bronchus in which the material has lodged and creates a favorable medium for anaerobic culture. Aspiration is most likely to occur with loss of the pharyngeal reflexes.

If we assume the correctness of this hypothesis, we may concede the existence of optimal conditions for the development of pulmonary abscess following the extraction of teeth. The clinical importance lies in the fact that no efforts directed toward meticulous hygiene as a preextraction precaution are made at the present time. It is safe to state that most teeth which are extracted are surrounded by subgingival pockets harboring débris, anaerobic organisms, and calculus (tartar). These elements are necessarily dislodged in the operation and are frequently carried back by the saliva and blood into the pharyngeal region. Furthermore, a unilateral dulling of the pharyngeal reflex is effected by two commonly employed nerve-block injections: namely, those for the inferior dental and

the anterior palatine nerves. Therefore, easy passage of such material into the trachea and aspiration into the bronchial tree are constant possibilities.

It is also noteworthy that not infrequently an ooze of blood and an increased flow of saliva continue for a time after operation. The opportunity for aspirating septic material in this interval, particularly during sleep, is obvious.

If the aspiration of particulate substance occurs during and after operations in the oral cavity with the frequency indicated by these data, it is evident that certain factors must operate fairly consistently to abort or inhibit infection. Probably the most important reaction of this type is the tendency of the lungs to rid themselves of foreign material, chiefly through coughing. The normal aeration of the lungs may be important in inhibiting anaerobic growth. Likewise the quantity and the virulence of the infected material, as well as the duration of their contact with susceptible tissues, are modifying factors.

THE TIME FACTOR IN THE DEVELOPMENT OF PULMONARY ABSCESS

The twelve patients whose cases are reported in detail developed pulmonary abscesses following dental operations after varying time intervals, and, while Moore[13] recognizes the possible lapse of 150 days after operation before symptoms are noted, there must be a great deal of doubt as to the reliability of such an assumption. The first clinical manifestations of abscess of the lung are often noted within a few days following operation and in the experiences of my colleagues continue in a fairly orderly sequence. They are described by Neuhof and Wessler[29] as follows:

"A definite period of incubation, in which no obvious symptoms can be said to exist. . . . The initial symptoms are feverishness and chilliness or an actual chill. . . . They are usually followed by pain in the chest. . . . Circumscribed tenderness to pressure is associated with the pain. . . . Cough usually begins after the onset of pain and is unproductive at first. A foul odor on coughing may be noted. Expectoration of foul pus begins suddenly, and at a characteristic time. . . . This is the tenth to twelfth day after the onset."

This sequence of symptoms with a correlation of the pathology of pulmonary abscess is similarly and more minutely described by Touroff and Moolten.[30] It may be conceded, however, that an accurate chronologic record is not always obtainable particularly in those cases first seen late in the course of the disease and that therefore a certain margin of error may exist. Furthermore, the clinical manifestations may be atypical. For example, when the pulmonary abscess is largely shut off, the foul odor to the breath on coughing may be the sole evidence of the infection for an extended period. While there is no set maximum interval following operation before the onset of symptoms, it is fair to

assume that the two-week interval represents the usual limit. A true etiologic relationship becomes doubtful if symptoms begin several weeks or longer after the assumed cause.

Although in three additional cases among those studied at Mount Sinai Hospital the histories suggested that dental operations were etiologic factors, the cases to be reported are limited to those in which the onset is classic and the relationship of the dental operation clear-cut. The fact is emphasized that in none of these cases was there evidence of aspiration of teeth by roentgenogram, by bronchoscopy, or at operation on the abscess of the lung.

REPORT OF CASES

CASE 1.—Hospital No. 308398. V. S., aged fifty-one years, was admitted on Nov. 5, 1929, with the following history: The right maxillary second premolar was removed under nitrous oxide anesthesia on Oct. 14, 1929, the entire tooth being displayed by the dentist immediately after operation. Aside from a slight coughing spell that night, the patient felt well until the third day at 5 P.M., when a chill lasting about one hour occurred, accompanied by pain in the right chest posteriorly. Malaise and a temperature of 101.5° F. developed the next day, followed shortly by slight cough with mucoid expectoration. This condition remained unchanged until admission, when there was a marked exacerbation of symptoms, inability to eat or sleep, and high temperature.

The patient exhibited an extremely unhygienic mouth with large tartar deposits and bleeding gums. Studies in the hospital indicated the diagnosis (confirmed at operation) of foul pyopneumothorax on the basis of a ruptured abscess of the lung located in the left lower lobe.

CASE 2.—Hospital No. 318726. T. P., aged thirty-two years, was admitted on Sept. 26, 1930, eight weeks after the left maxillary second molar had been removed under local anesthesia. Although previously well, the patient felt tired and irritable and had occasional headaches for the week thereafter. Then she experienced sudden severe pain in the left shoulder at the angle of the scapula, the pain increasing with respiratory effort. This persisted for two days and disappeared with the onset of malaise, body aches, and fever ranging from 103° to 104° F. At the end of five weeks a cough developed; there was a foul odor to the breath and expectoration of foul greenish sputum in considerable amounts.

The mouth and teeth were in moderately good condition. At operation a putrid pulmonary abscess was found in the posterior portion of the left lower lobe.

CASE 3.—Hospital No. 326350. M. E., aged fifty-five years, was admitted on May 25, 1931. The mandibular left central incisor had been removed about three weeks previously under local anesthesia. Several hours afterward the patient experienced a severe shaking chill lasting several hours. There were no further symptoms until four days later when malaise with generalized aches and pains and a temperature of 103° F. developed. This was followed rapidly by the onset of a harassing cough productive of whitish nonodorous sputum which rapidly became more frequent and troublesome. After three weeks the sputum became greenish in color and foul in odor.

The patient's maxilla was edentulous, the lower teeth unclean and pyorrheal. The history, physical findings, and roentgenograms (verified by bronchoscopy) indicated an abscess of the lung in the left upper lobe.

CASE 4.—Hospital No. 329731. G. W., aged twenty-four years, upon admission Sept. 3, 1931, gave the following history: Because of infection the maxillary right first molar was removed under local anesthesia three and one-half weeks previously. The first symptom of her present illness occurred ten days later in the form of a marked throat irritation accompanied by a foul odor to the breath. This was shortly followed by a cough and the expectoration of foul, yellowish sputum. There was no fever or dyspnea at this time. Three weeks after the extraction a severe but dull pain was experienced in the anterior right chest at the level of the fourth rib, this area being tender to touch. Simultaneously, malaise and fever developed.

Examination of the mouth disclosed teeth and gums in an unhygienic condition. Thoracotomy was performed demonstrating a putrid abscess of the lung in the right upper lobe.

CASE 5.—Hospital No. 330020. I. G., aged twenty-six years, stated that in January, 1931 (nine months before admission) the maxillary left first molar had been removed under local anesthesia. A week later a sticking, drawing pain which was intensified by inspiration developed in the left axillary region. This lasted for a day and was accompanied by malaise. There was an interval of two weeks in which the patient was apparently well. Then suddenly he began to cough and expectorate quantities of foul-smelling, yellowish sputum, occasionally blood-streaked. These symptoms persisted at the time of admission.

The mouth was in fairly clean condition, a small amount of tartar being adherent to the necks of the lower anterior teeth. The bronchoscopic finding, supported at operation, was putrid abscess of the lung in the lateral part of the left upper lobe with fibrosis and retraction of the mediastinum to the left.

CASE 6.—Hospital No. 343313. M. G., aged twenty-nine years, presented himself at the hospital Sept. 11, 1932, with an extensive fluctuating swelling involving the left mandible and submaxillary triangle, associated with an alveolar abscess of the mandibular left third molar. Nitrous oxide oxygen anesthesia was administered to permit incision and drainage. Shortly after induction the patient became excited and exhibited generalized twitchings. Suddenly a foul odor was noted. The cause of this was found in a spontaneous intraoral rupture of the abscess, the mask containing a quantity of pus. An extraoral incision was made. The patient became cyanotic and exhibited convulsive movements with labored breathing; the pulse was of poor quality. Recovery from this condition was gradual following discontinuance of anesthesia and administration of 5 per cent glucose and saline.

The patient was readmitted three and one-half weeks later. On the sixth day following the operation he had experienced marked pain in the right suprascapular region, which was aggravated by breathing. Ten days after this a cough developed, accompanied by expectoration of foul material. Foul breath and sputum persisted, associated with malaise and loss of 13 pounds in weight.

The mouth was in a fairly hygienic condition; the mandibular third molar was broken down and carious. Roentgenograms indicated an abscess of the lung in the axillary portion of the right upper lobe. Thoracotomy and pneumonotomy were performed, and the abscess roof was excised. (It is of interest in connection with the location of the abscess that the patient had reclined on the right side during anesthesia.) This case has been reported in detail by the writer.[31]

CASE 7.—Hospital No. 344443. H. B., aged forty-six years, was admitted on Oct. 11, 1932. Nine months previously he had suffered from chills and fever followed by a cough productive of a grayish sputum which was not foul. The expectoration continued for several months, diminishing in frequency and amount. He attended the clinic for several months thereafter.

Four weeks before admission sixteen teeth were removed under local anesthesia. Two weeks later the present illness began suddenly with chills and fever, followed in three days by a harassing productive cough, which was accompanied by sharp pain in the upper right anterior chest radiating to the axilla. The character of the expectorated sputum changed at this time, acquiring a bitter taste and a particularly foul odor.

The patient was edentulous. At operation multiple chronic abscesses in the right upper lobe of the lung were found.

CASE 8.—Hospital No. 348978. L. W., aged twenty-eight years, was admitted on March 4, 1933. Fifteen days previously the maxillary right first molar had been removed under local anesthesia. There was very slight bleeding from the socket.

Twenty-eight hours later she experienced a sudden sharp knifelike pain over the lower posterior area of the left axilla. The pain became constant, increased in severity, and was intensified by respiration. It continued for seven days, and although it had eased considerably at this time, twinges were still felt upon deep inspiration.

Five days prior to admission a severe paroxysm of coughing occurred, followed by continued fits of coughing of varying degree. The cough shortly thereafter became productive of foul sputum alternating between grayish yellow and white in color. The breath was continuously foul since then. There was no hemoptysis, elevation of temperature, chills, or malaise. The teeth and gums were in excellent condition.

The customary studies, supported by bronchoscopic findings, established the existence of an acute putrid abscess in the left lower lobe of the lung.

CASE 9.—Hospital No. 352637. M. W., aged fifty years, was admitted on May 27, 1933. Six teeth had been removed under nitrous oxide anesthesia sixteen days before. Within a few hours several prolonged chills occurred, followed by profuse perspiration.

Two days later the patient was seized with sudden sharp agonizing pains in the left axilla and back, intensified by motion. With this, a dry, hacking cough with little expectoration developed.

Four days before admission the pain and dyspnea increased, and a foul odor to the breath was noted. On admission the patient was acutely ill with a temperature of 102.2° F.

The remaining teeth were in very poor condition and were encrusted with tartar. The gums were markedly inflamed.

A pyopneumothorax was disclosed at operation, evidently due to a ruptured abscess of the left upper lobe of the lung.

CASE 10.—Hospital No. 353322. W. F., aged forty-four years, was admitted on June 13, 1933. Ten weeks before, the maxillary left canine had been removed under local anesthesia, and his present illness began two weeks later. At that time he felt quite ill, had fever, and suffered chills.

Pain developed in the left lower chest anteriorly and with it night sweats and a cough. A few days afterward the cough became associated with expectoration of large amounts of sputum which was neither foul nor bloody.

The teeth were stained and exhibited large deposits of tartar; the gums were inflamed.

Confirming the impression gained by the studies in the hospital, bronchoscopy disclosed an abscess in the left lower lobe of the lung, with a partial rupture into the pleural space and pyopneumothorax.

CASE 11.—Hospital No. 357703. T. H., aged sixty years, was admitted on Oct. 3, 1933. Because of pyorrhea all of the patient's teeth had been removed under local anesthesia over a period of several months. The mandibular left second molar was the last to be extracted—four months before admission. Apparently as a complication of the extraction, a retromolar abscess developed; this was incised and drained under local anesthesia.

The present illness began one week later with chills, fever, and pains in the lower right chest, anteriorly and posteriorly. A bad taste and a foul odor to the breath were noted. The patient began to cough, expectorating large amounts of foul-smelling greenish sputum. Three weeks before admission a severe hemoptysis occurred.

The patient was edentulous.

Bronchoscopy revealed pus discharging from the apical branch of the right lower lobe and left lower lobe, as well as from the paravertebral branch of the left lower lobe. In view of the patient's age, general condition, and the extent of the disease, surgical intervention was held unwarranted.

CASE 12.—Hospital No. 346613. C. K., a dental hygienist aged twenty years, was admitted on Dec. 10, 1932, with a history of intermittent cough for a period of over a year. Six weeks before admission she underwent a thorough prophylactic treatment of the teeth involving the removal of deposits of tartar from underneath the gums, which were inflamed. Three weeks later she became conscious of a foul odor to the breath and developed a pain originating in the right lateral back and radiating to the suprascapular area. After a week there was a severe and continuous exacerbation of the cough with a production of foul sputum. After that she suffered daily chilly sensations with a temperature ranging up to 103° F. During the three days prior to admission there were several hemoptyses.

The diagnosis reached was of a putrid abscess in the upper lobe of the lung, with tuberculosis of the right lower lobe and of the larynx.

COMMENT

Local anesthesia was employed for the dental operations in eight cases, nitrous oxide in three, and none in one. The fact may be stressed that in five cases teeth were removed from the posterior part of the maxilla where the customary procedure is to block the anterior palatine nerve which affects the reflex mechanism of a portion of the soft palate (Cases 2, 4, 5, 7, and 8). In two instances the mandibular and lingual nerves were blocked, reducing the sensitiveness of the anterior fauces and pterygomandibular space (Cases 3 and 11). These data may be interpreted as suggesting a relationship between aspiration and the obtunding of reflexes in the posterior part of the mouth by novocaine. The history and appearance of the mouths indicated lack of oral hygiene in most patients at the time of the dental operation. If the aspiration of tartar may be regarded as a possible etiologic factor in abscess of the lung (and there is much evidence supporting this assumption), Case 12 presents a reasonably typical history.

PREVENTIVE MEASURES

The reduction of the incidence of pulmonary abscess following dental operations probably depends entirely on obviating aspiration. The

nature of the material which, if inhaled, may produce abscess of the lung is varied, the most likely particles being fragments of tartar dislodged during operation. Detached bits of soft structures, such as chronic inflammatory tissue, blood clot, bloody saliva and pus, may also be mentioned. The assumption is that such material bears anaerobes of the type that produces abscess of the lung. It must also be emphasized that aspiration may occur either in the course of the operation or in comatose states immediately following operation if blood or fragments remain in the mouth.

The most obvious preventive measure is a meticulous toilet of the mouth, particularly of the teeth to be extracted, before operation. This should include high pressure irrigation of the interdental spaces, the use of dental floss, and especially the painstaking removal of tartar from about the teeth to be extracted. The use of sodium perborate as a preventive mouth wash is probably valuable because of its oxygenating properties. The amount of novocaine to be injected should be the minimum actually required for anesthesia, as the injection of surplus amounts, particularly into the posterior palatine foramen, interferes to some extent with the cough reflex mechanism. The entire gingival margin around the tooth to be extracted should be actually cauterized by heat after anesthesia has been induced. For reducing the hazard of aspiration during operation, it is ideal to have the patient prone, even under local anesthesia. Morning extractions are preferable because the maximum time is allowed for complete control of hemorrhage before the patient ordinarily sleeps. For the same reason, the use of hypnotics to control pain is to be avoided the first day. Careful attention should be paid to the control of postoperative bleeding, both by proper management of the wound and instructions to the patient. The inhibiting effect on anaerobic activity of an oxidizing agent, such as sodium perborate, hydrogen peroxide, or potassium permanganate as a mouth wash, is pronounced enough to warrant its routine use after extraction.

Finally all patients should be seen three weeks after operation in order to check up on the interval history with reference to symptoms of pulmonary abscess. This follow-up is almost axiomatic and involves nothing revolutionary or complicated. Until more information on the subject accumulates, the follow-up system offers a logical starting point in prevention. Many of those who perform tonsillectomies were in the habit of saying that they never saw a posttonsillectomy abscess of the lung in their practice—until they employed a follow-up system. Perhaps the same experience may be in store for those who perform extractions. In any event the follow-ups and the study of a large amount of material in this field will furnish the evidence concerning the question of mouth hygiene before and after extraction.

SUMMARY AND CONCLUSIONS

1. Twelve of seventy consecutive and unselected patients with pulmonary abscess gave histories strongly suggestive of causes traceable to dental operations. In view of this high percentage, abscess of the lung is apparently a more common complication of dental surgery than is generally assumed to be the case.

2. Pulmonary abscess may occur after tooth extraction without the aspiration of gross material, such as a tooth or filling. Gross aspiration did not occur in any of the cases reported in this series. More cases followed the use of local than of general anesthesia. This is probably accounted for by the fact that today local anesthesia is the method of choice in dental surgery.

3. Thorough investigation of the dental history in cases of abscess of the lung may reduce the percentage of cases formerly grouped as pulmonary abscess of unknown origin.

4. Clinical and pathologic evidence point to aspiration as the usual mechanism in the production of putrid pulmonary abscess. The anesthesia and other factors associated with tooth extraction offer optimal conditions for the aspiration of infected particulate matter.

5. There is evidence to suggest the association of a diminished or inactive reflex during dental operations with the occurrence of abscess of the lung.

6. The prevention of pulmonary abscess following dental procedures may depend on the recognition and elimination of the hazards of aspirating infected particulate material dislodged during such procedures.

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THE SYMPTOMATOLOGY OF PUTRID ABSCESS OF THE LUNG*

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INTRODUCTION

DURING recent years much has been written of the various aspects of putrid abscess of the lung. Although the etiology, pathology, diagnosis, and treatment of this fairly common disease have been discussed extensively, comparatively little attention has been paid to the subject of its detailed symptomatology. It is our feeling, therefore, that some form of tabulation and analysis of symptoms, especially early symptoms, is timely. In many instances a careful case history proves to be the most important factor in clarifying the diagnosis and differentiating it from a number of other conditions with which it may be confused. By the same means it may be possible also to localize the lesion more exactly and to establish its prognosis.

Putrid abscess is a primary disease of the lung, presenting characteristic pathologic and clinical features which distinguish it from other types of pulmonary affection, putrid and otherwise. The description to be presented, of the typical clinical course of the disease and some of its more common variations, is based on a study of the case records of 158 patients with putrid abscess of the lung who were under treatment by the Thoracic Group of the Mount Sinai Hospital during the years 1928 to 1933, inclusive.

ETIOLOGY, PATHOGENESIS, AND PATHOLOGY

As a basis for an explanation of some of the clinical manifestations which occur, we shall describe in outline the etiology and pathology of this disease in accordance with the views which we share with others of the Group.[8] We consider putrid lung abscess to be a disease produced by aspiration into the bronchi of certain anaerobic microorganisms[2] that cause gangrenous infection. The original seat of these organisms is almost always in the mouth and pharynx, especially about the teeth and gums, and in the tonsils. The mechanism which causes such organisms to enter the bronchi varies considerably. Very often aspiration occurs during or following operations upon the mouth or throat. Thus about 25 per cent of our cases followed tonsillectomy, and about 12 per cent

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followed extraction of one or more teeth. Surgical drainage or spontaneous rupture of abscesses of the jaws, tonsils, or salivary glands was responsible in some cases. It is noteworthy that in our series operations upon the nasal passages and accessory sinuses were never followed by lung abscess, possibly because of different bacterial flora in these regions. In many cases in which no direct cause as described above was found, an indirect cause was often responsible, particularly the loss or blunting of the normal protective reflexes of the pharynx and larynx (i.e., gagging and coughing). This occurred after the application of cocaine locally to these regions or as the result of complete loss of consciousness. In many of the latter cases, unconsciousness was associated with irregular or depressed respiration and with vomiting. Thus a number of cases followed ether anesthesia, alcoholic stupor, diabetic coma, morphine overdosage, carbon monoxide poisoning, submersion, epileptic attacks, and perhaps even deep sleep. In seventy-three cases, comprising nearly half the entire series, no clue to the etiology was elicited, even with the most careful questioning. One of our colleagues[11] has found in many of these "idiopathic" cases, pyorrhea alveolaris associated with extensive dental deposits of tartar. His studies, which are soon to be published, strongly suggest the possibility of the aspiration of infective material from these sources during sleep.

The mass of aspirated infective material is usually of small size and becomes arrested only within a terminal bronchus or bronchiole of fine caliber. At that point it initiates an intense necrotizing inflammatory reaction which usually includes the tributary bronchioles and the pulmonary parenchyma which the latter supply. The lesion is therefore characteristically situated in that portion of the lung which lies only a short distance beneath the surface of the visceral pleura. An early reaction in the overlying visceral pleura, which rapidly involves the adjacent parietal pleura, invariably occurs. A certain number of abscesses lie adjacent to an interlobar fissure or to the mediastinal surface of the lung. The latter types of abscess are therefore also primarily superficially situated, but in roentgenograms may give the impression of being central. In our opinion the distinction made by some observers between superficial abscess and deep or central abscess of the lung is based upon erroneous interpretations of roentgen films.

The pathologic lesion of the first stage of putrid abscess consists of primary tissue gangrene with surrounding intense suppurative pneumonitis. The gangrene or putrid necrosis affects the entire parenchyma of the involved area including the bronchioles and blood vessels. A line of demarcation, which is quickly formed about the gangrenous focus, separates the latter from the surrounding inflamed lung tissue. As the necrotic tissue softens and liquefies, a cavity is produced. The material filling this cavity is thus composed of sloughing lung tissue and pus having an extremely foul, pungent odor.

As the process of tissue breakdown proceeds, one or more bronchi of fair size usually become eroded, thus providing natural drainage and aeration of the cavity by way of the bronchial tree. In some cases the entire putrid slough separates and is eliminated in this way with spontaneous cure of the abscess. In the majority of cases, however, bronchial drainage and aeration are inadequate, and part of the bacteria-laden slough fails to separate from the wall of the cavity. As a result, the infection is perpetuated. In the majority of instances, therefore, the lesion becomes subacute and either remains localized and more or less quiescent for weeks or months with occasional peripheral flare-up, or else insidiously invades the adjacent lung tissue. There may be direct spread of gangrene and of suppuration into the surrounding lung tissue, or "spill-over" may occur through the draining bronchus into adjacent normal bronchi. Ultimately a multilocular abscess or multiple contiguous abscesses are produced; the adjacent pulmonary parenchyma becomes diffusely inflamed and fibrotic, and the bronchi become dilated. This constitutes the chronic phase of the disease. It is usually reached in three or four months and, once established, practically never subsides spontaneously.

The secondary changes in the lung parenchyma including the bronchiectases may in time increase to such an extent that they completely overshadow the original underlying abscess. If the patient does not come under observation until late in the course of the disease, the condition may be confused clinically and even pathologically with primary bronchiectatic disease. In many of these cases it may be possible to clarify the diagnosis by a careful review of the details of the patient's earliest symptoms.

Early pleural reaction is a constant finding in all cases of lung abscess. Firm fibrous adhesions are usually formed between the two layers of the pleura at the site of the lesion. In some cases, however, these adhesions are inadequate to prevent a later perforation of the abscess into the pleura, with the production of putrid pyopneumothorax, either localized or generalized. In a few cases the primary gangrenous pulmonary lesion rapidly involves the overlying visceral pleura, and perforation into the pleural cavity occurs in the earliest phase of the disease before drainage into the bronchi has become established. In such cases the presenting clinical features are those of an apparently primary putrid pyopneumothorax rather than those of lung abscess.

CLINICAL MANIFESTATIONS

A. *Clinical Course During Primary Stage.*—In the typical case of putrid lung abscess the symptoms of onset are mild. The first symptoms usually appear within a few days or a week after infection of the lung occurs. The patient begins to have a dry irritating cough, then chilli-

ness followed by slight fever, and soon afterward stabbing pain in the chest. In a short time he begins to expectorate small amounts of mucopurulent sputum.

These rather mild symptoms become somewhat worse. Fever may reach 102° or even 103° F., but rarely more, and is irregular and attended with sweats. The patient feels mildly prostrated, complaining of loss of appetite, weight, and strength. During this time the clinical picture simulates that of an ordinary acute respiratory infection. Before the end of the second week of illness, however, the first specific evidence of the disease appears in the form of foul odor of the breath and sputum. Usually by this time a certain amount of blood has been seen in the sputum on one or more occasions in the form of either fine streaks or small clots.

Physical signs at this stage are usually meager. A small area of localized dullness and râles may be detected at the site of the chest pain, but rarely are signs of cavitation found. Roentgen ray examination suggests merely the presence of a patch of pneumonic infiltration.

From this time on, in most cases, the amount of sputum increases considerably and becomes much more foul. With this increased spontaneous drainage and ventilation by way of the bronchial tree, the patient begins to feel better, fever becomes less, and pain in the chest disappears. The roentgenogram for the first time now discloses a cavity with a fluid level surmounted by air, which is characteristic of lung abscess.

As drainage and ventilation continue, the amount of sputum, the intensity of the foul odor, fever, and toxemia usually lessen. The patient may now be considered to have reached a crucial point, so far as he either may go on to spontaneous cure or may merely enter a period of temporary remission. In connection with the use of the term "cure" as applied to lung abscess, it must be emphasized that clinical improvement alone is not an adequate criterion. Disappearance of all pulmonary infiltration in the roentgen film, in addition to disappearance of all clinical manifestations, is the only reliable indication of complete recovery. Employing this criterion we have found that only 23.5 per cent of our patients went on to spontaneous cure. On the other hand, the mortality of the primary stage of typical lung abscess is exceedingly low. Death at this stage occurs usually only in markedly debilitated subjects, particularly in the presence of diabetes or senile cachexia. In general, the primary stage of putrid lung abscess is marked by the mild character of its symptomatology and low degree of toxemia.

B. *Stage of Remission: Relapse.*—The relapsing character of putrid lung abscess apparently is not yet sufficiently appreciated. Emphasis on this point must be made particularly in the case of patients who are discharged as improved. In some of these patients all symptoms cease except a slight dry cough; their lost weight is regained, and their feeling

of well-being returns. In spite of the patient's clinical improvement, however, the roentgen film fails to demonstrate the disappearance of infiltration, although the cavity may appear to be smaller or may even not be visible. The remission may last no more than a few days or may continue for weeks, depending in all likelihood upon the degree of drainage and ventilation of the cavity. Relapse is ushered in by recurrence of fever, chest pain, hemoptysis, and profuse foul sputum. Roentgenograms show corresponding increase in density and size of the infiltration. The cavity may not become apparent again within the area of infiltration until drainage improves. A secondary anemia develops fairly rapidly, and the skin assumes a peculiar waxy translucent pallor. Clubbing of the fingers and toes is often pronounced. Even in this phase a few patients undergo spontaneous recovery, but much more slowly than in the primary acute stage. At the end of the third or fourth month, fibrous induration and bronchiectases are fairly marked, and the outlook for spontaneous recovery is extremely remote. This we may now properly term the chronic phase of lung abscess.

C. *Chronic Stage.*—The clinical manifestations of the chronic stage of putrid lung abscess vary with the progress of both gangrenous suppuration and secondary fibrosis. In one type of case there are periodic acute exacerbations, with fever, chest pain, and increase in foul sputum. In another type of case there is little change in the general condition; the patient remains fairly comfortable for a long period of time but continues to expectorate large amounts of foul sputum. In a third variety the anaerobic infection apparently becomes more or less burnt out, and the foul odor disappears; a copious nonodorous expectoration persists, however, and the clinical picture becomes that of bronchiectatic disease.

Regardless of type, the chronic case of lung abscess which is untreated is destined to a course of progressive deterioration. In our experience the average duration of life in adults is less than three years. The hopeless prognosis of such cases actually parallels that of cancer. Death may result from spread of the disease within the lung itself, such as acute gangrenous bronchopneumonia, by direct spread or "spill-over," perforation into the pleura or into the pericardium, or fatal pulmonary hemorrhage. Cachexia associated with long-standing toxemia and, in particular, with amyloid disease of the viscera, may accelerate the fatal outcome. Metastatic abscesses cause death in some instances; these abscesses, like the primary pulmonary focus, are usually putrid. In these cases autopsy may reveal contact involvement of one or more small veins lying near the abscess in the lung or within its wall; these veins contain purulent thrombi from which, in all probability, infective emboli become liberated.[1] The more common sites of metastatic putrid abscess are the brain, kidney, and long bones. In five cases reported by one of us in a separate communication,[10] death occurred as a result of suppurative

pylephlebitis with multiple putrid liver abscesses. The infection of the portal vein in each of the five cases was traced directly to a metastatic putrid abscess of the spleen. As far as can be determined this is the only mechanism of putrid infection of the liver complicating lung abscess. Direct extension of putrid lung abscess through the diaphragm into the liver has never been observed by us, nor have authentic cases been reported in the literature.

VARIATIONS IN SYMPTOMATOLOGY

In the individual case of lung abscess, variations from the usual course often occur, and at times are so pronounced that they completely dominate the clinical picture. In studying an individual case, the clinician must carefully note, for purposes of comparison, certain important diagnostic features: namely, (1) the type of onset, (2) foul odor, (3) chest pain, and (4) bleeding.

1. The typical onset, as we have already stated, is mild, as in grip. In a certain proportion of cases (about 15 per cent) the onset is, however, rather severe as in influenza or lobar pneumonia, and begins with a shaking chill. These patients are often classified as cases of postpneumonic lung abscess, on the assumption that their primary illness was lobar pneumonia. Actually a lung abscess has been present from the beginning.* Surprisingly, almost all cases with hyperacute onset studied by us belonged to the idiopathic group, i.e., without known etiology. Their further course was similar to that of any other type of putrid lung abscess.

Certain individuals in this series passed through the primary acute stage of their disease with hardly the slightest indisposition, and were puzzled by the persistence of cough and bad smelling sputum. These were generally mild infections. Roentgenograms in such cases revealed relatively small areas of infiltration casting a shadow of low density but with distinct cavitation. Many of these healed spontaneously.

In young children the onset is often very atypical. This, however, may be more apparent than real because of the tendency of children to swallow their sputum and not to complain of mild symptoms. As a result, the first stage of the disease may pass unnoticed by the parents, and the important symptoms become manifest only after the chronic stage has become established, weeks or months after the onset.

For the same reason, a young child or infant may run a septic fever for weeks with almost no other obvious symptoms of illness until examination discloses putrid empyema secondary to an unrecognized rupture of an acute lung abscess.

*One must take care to distinguish primary putrid lung abscess with pneumonia-like onset from the various forms of necrosis of lung[3,5,6] tissue occurring in severe lobar pneumonia, especially in diabetic persons. The latter type of lesion is probably caused by vascular injury and is *nonputrid*. In contrast to putrid lung abscess it follows a relatively benign course, often without symptoms, and resolves spontaneously.

2. The *foul odor* is the characteristic stigma of putrid bronchogenic lung abscess. It is present at one time or another in almost every case. The exceptions are those cases in which the lung abscess almost at onset ruptures into the pleural cavity. In such cases there is very little cough, no sputum, and no foul breath; and only the extremely severe pain in the chest indicates the presence of a virulent empyema.

While foul odor is always present in the ordinary case of uncomplicated lung abscess, it is not always detected. It may at times be so evanescent that its presence can be determined only on bronchoscopy. More often the patient has considerable sputum without odor, and only after a paroxysm of coughing will the odor be detectable in the breath. (Because of its great importance in diagnosis and prognosis, the physician must not be too fastidious to make a careful study of the patient's breath during exhalation and immediately after coughing.) In some patients the foul odor of breath and sputum increases gradually from day to day, and the disease in general pursues a rather sluggish course. In such cases, small particles of foul-smelling slough from the abscess cavity may be coughed up from time to time as minute grayish black pellets easily separated from the sputum; microscopically such particles appear to be composed almost entirely of bacteria, chiefly gram-negative bacilli. On the other hand, certain patients first expectorate their foul sputum in one enormous gush of stinking fluid, reddish black or grayish black in color. In such cases considerable symptomatic improvement usually follows for a varying period.

It is of more than passing interest to note that a large number of patients with lung abscess give the time of first occurrence of foul sputum as the twelfth to fourteenth day after infection has taken place. Some variation from this is seen in a number of cases, and a few patients have foul sputum as early as the fourth day. The latent period probably represents the time required for the earliest separation of the putrid slough from its wall of demarcation in the abscess permitting egress of foul material into open bronchi; its two-week duration may signify something of the biologic characteristics of the organisms which cause the lesion.

Even after foul sputum has become established, it may be inconstant and appear only at certain times of the day. Of greater importance is the continued absence of foul odor early in the course of the disease. Its significance depends entirely upon associated clinical phenomena. One of the most serious complications responsible for the continued absence of foul sputum is rupture of the abscess into the pleural cavity. One should therefore view with alarm a disappearance of foul odor if it is accompanied by abrupt decrease in the volume of sputum expectorated, intense and diffuse chest pain, and extreme toxemia. On the other hand, if disappearance of foul odor is accompanied by moderate decrease in sputum, moderate chest pain, and slight or moderate fever, one may

place a relative value upon these symptoms as indicative of the degree to which drainage of the abscess is being hindered by the partial shutting-off of its draining bronchus. In the latter case one may also anticipate spontaneous remission of symptoms after four or five days, at which time the patient usually expectorates a large amount of foul sputum. In some instances the observer is uncertain whether or not he is dealing with incipient or slow perforation of an abscess or merely with "shutting-off." In such cases careful examination at frequent intervals, supplemented by roentgenography, is always necessary. The obverse of this picture is cessation of foul odor together with most other symptoms. The uninitiated observer under such circumstances may be led to believe that early cure is imminent; whereas the further course will prove this to be but a temporary remission. In such instances, likewise, successive roentgenograms are indispensable.

The foul odor of lung abscess is such an important element in the clinical picture that the observer must school himself thoroughly to differentiate this odor from other offensive odors of the breath and sputum. Pyorrhea alveolaris and ozena produce a very disagreeable odor of the breath. Sputum also acquires an offensive odor after remaining for several hours in a closed receptacle. In bronchiectatic disease of advanced type with widely dilated bronchopulmonary sacculations, the patient often develops a certain insensitivity to cough-producing stimuli so that large amounts of purulent secretion accumulate in the bronchi and are retained for many hours. As a result the stagnant sputum often acquires a rancid offensive odor which may simulate that of putrid lung abscess. The quality of the odor of putrid lung abscess is, however, altogether distinctive because of a certain characteristic acridity or pungency quite apart from the actual stench.

Certain lesions within the lung may at times become secondarily infected with putrefactive anerobes, especially in patients with badly diseased teeth and tonsils. This complication is easily recognized clinically by the presence of the distinctive foul odor of putrid infection. Putrid superinfection is particularly frequent in the later stages of bronchiectatic disease but is also seen at times as a complication of bronchial obstruction by tumor or foreign body, in hematoma of the lung resulting from severe contusion of the chest or in hemorrhagic infarcts such as occur in heart disease. Although their early courses are quite different from that of primary lung abscess, such patients are subject to the same vicissitudes as those with lung abscess, including perforation of the putrid focus into the pleural cavity.

In some cases one can deduce from the amount of stench alone, the condition of the abscess and the patient's prognosis. Generally speaking, a very marked stench usually signifies advanced disease with extensive secondary putrid bronchiectasis. In markedly debilitated individuals, such extreme foulness of the sputum may occur early in the disease as

a manifestation of rapidly advancing gangrenous bronchopneumonia of one or both lungs. In such cases death usually occurs in a short time. On the other hand, in certain early cases of lung abscess with an extremely foul odor, the outlook paradoxically may be excellent. In such cases roentgenograms show a single large lesion with rapid sloughing away of the gangrenous portion from the surrounding healthy lung, particularly after surgical drainage. In such event a clean hole is left in the lung which is quickly filled by compensatory expansion of the surrounding alveoli.

3. Of great importance from the standpoint of diagnosis and treatment is the *chest pain* which is present in the first stage of almost every case of lung abscess. In nearly one-third of our cases, pain was actually the first presenting pulmonary symptom, even preceding cough. Pain is caused by the pleural reaction that develops over the abscess in its earliest stages, and in most cases is localized to a single area of the chest wall which overlies the site of the pulmonary lesion. If tenderness is also present in the intercostal space, one can be even more certain of the localization. To the surgeon this is of the utmost importance in deciding where to drain, since the densest adhesions are usually found at this point. In nearly every case which we have observed at operation, regardless of how early operation was performed (in one case on the twelfth day), adhesions were noted overlying the site of the abscess at its most superficial point. In cases with little or no pain (which are very rare) one may expect to find few or no adhesions at operation. In such cases primary one-stage drainage of the abscess is contraindicated, and a two-stage procedure is necessary to protect the free pleural space from contamination. In the majority of instances the adhesions are firm and sufficient in extent to permit drainage in one stage, provided the surgical approach is made at the proper point.

The pain is usually described as stabbing in type and of moderate severity. In a certain number of patients, pain is of great severity. What is more important than severity of pain, however, is its duration. When pain of severe character is present steadily for several days or a week, one must look for signs of perforating lung abscess. If the intensity of the pain also increases and a wider area of the chest wall becomes affected, free perforation must be anticipated. As a rule, the resulting empyema is encapsulated rather than diffuse, and the prognosis after surgical drainage is fairly good. Physical signs now are of considerable help in diagnosis and localization since they are well defined in contrast to the signs of uncomplicated lung abscess.

Mildness of chest pain or its absence may foreshadow spontaneous cure of the disease. In such cases the fever also is mild, sputum not very profuse or very foul, and the shadow in the roentgenogram of small size and low density.

One must be on guard against atypical radiation of pain. This is particularly important when the abscess faces the diaphragm. Pain, under such circumstances, may be referred either to the front or to the back of the lower chest, or may be referred only to the shoulder and neck along the trapezius ridge.

4. *Bleeding* is such a regular symptom of lung abscess, occurring in practically every case, that it is not surprising that many cases of lung abscess at first are confused with tuberculosis. Actually, bleeding is far more frequent in lung abscess than in either tuberculosis or cancer of the lung. It may occur as profuse hemoptysis but much more often consists in mere blood-streaking or the presence of a few clots. Very often hemoptysis ushers in the reactivation of an abscess after a period of quiescence and apparent improvement and probably indicates the spread of gangrene into fresh lung tissue. A patient with lung abscess may bleed to death from sudden tremendous hemoptysis, which is a comparatively rare occurrence in tuberculosis. Death also may occur indirectly from bleeding, either by the aspiration of air or of pus into an open vein. In the former case, death is immediate from air embolism and, in the latter, takes place after a varying period of time as a result of multiple septic metastases.

CONSTITUTIONAL SYMPTOMS

General symptoms are usually moderate, as already mentioned. During the primary stage and during exacerbations, fever ranges usually between 100° and 103° F., but in some cases is slight. In the pneumonic type of onset it may be high. Chilliness, sweats, malaise ,anorexia, loss of weight, weakness, and body aches are usual and vary with the intensity of the toxemia. Anemia develops rapidly even with slight hemoptysis. Leucocytosis varies with the activity of the disease and counts between 10,000 and 25,000 are not infrequent; higher counts suggest complications. Psychic changes are rarely noted except during severe toxic states or with metastatic brain abscess or air embolism. In the chronic stage of the disease, an interesting tendency to hyperactivity of tendon reflexes and clonus is observed.

Clubbing of the fingers and toes appears early in lung abscess and becomes marked as the disease progresses. It may be seen as early as the fourth week, a fact in striking contrast to its occurrence in tuberculosis in which several years elapse before clubbing is seen. The bony parts of the digits as well as the soft parts enlarge, and later the distal ends of the long bones and the lower jaw also. Roentgenograms may show periosteal production of bone as in periostitis, and the patient may complain of rheumatic pain in these regions, especially in the ankles and wrists. Other associated changes, such as thickening of the skin of the face with corresponding exaggeration of the natural wrinkles and a

mild seborrhea, are frequent. After surgical drainage of a chronic lung abscess, clubbing often becomes less yet persists in some degree, possibly because of the persistence of the complicating bronchiectases. Complete disappearance of clubbing is seen after cure of an acute lung abscess; its persistence in spite of treatment in such cases indicates a persistent, undrained focus of infection.

PHYSICAL EXAMINATION AND ROENTGENOGRAPHY

Physical signs of lung abscess are usually surprisingly few. In the primary stage, while the abscess is still solitary and circumscribed, a small patch of moderate dullness, moist râles, and bronchovesicular breath sounds may be discovered. In some cases even these are lacking, and it is impossible to locate the lesion except by roentgenography. Almost never are the classical signs of cavity noted in uncomplicated cases. For this reason, if an otherwise simple case presents extensive physical findings, one should either suspect a complication or reconsider the original diagnosis. The most important complication to be sought is perforation of the abscess into the pleura, the physical signs in such a case being generally those of encapsulated pyopneumothorax. In our experience this is the type of case in which the most perfect examples of the classical "signs of cavity" are found, viz., amphoric voice and whisper, and loud clicking râles. In the later stages of chronic lung abscess, physical signs in general are much more marked because of secondary pulmonary fibrosis and bronchiectases.

Roentgen examination is one of our most important aids in studying each case. For localization of the abscess, a lateral film is usually taken as well as the ordinary posteroanterior films, single or stereoscopic.

The earliest shadow resembles a patch of simple pneumonic infiltration and may be clearly seen very soon after the infection has been acquired. Rarefaction within the shadow begins shortly afterward, and progresses to form a definite rounded cavity. The beginning of cavitation usually corresponds to the first production of foul sputum and indicates the inception of spontaneous drainage and ventilation by way of the bronchial tree. Often the cavity appears and then quickly becomes invisible, as it refills with pus; it may be possible later to demonstrate it again by postural drainage. When the cavity has reached its greatest size, it is usually spherical, but may have a clover-leaf or dumb-bell shape. It rarely reaches the size of the full extent of the pneumonic shadow within which it occurs. The zone of infiltration which therefore remains as the wall of the cavity varies considerably in width. During the further progress of the disease, this wall of infiltrated lung tissue changes both in thickness and in density. In a case which is tending toward spontaneous cure, it becomes lighter and narrower until entirely absorbed or reduced to a fine streak of scar tissue. In cases which

progress, it expands and sooner or later presents other cavities, either large cavities caused by fresh gangrene or small bronchiectatic cavities. Lipiodol fills the latter type, but does not, as a rule, fill the cavities of the abscess proper; this is an important point in localization by this method since, in our experience, the bronchus which fails to fill with lipiodol usually corresponds to that leading from the abscess[4] Meanwhile the infiltration itself changes, assuming a dense fibroid character. It may become of such density that cavities are obscured, and the film then may suggest the presence of a tumor. More often, particularly if situated in an upper lung field, it may be confused with tuberculosis.

Discussion of special diagnostic methods is not within the scope of this paper. In passing, however, we refer to bronchoscopy[7] to emphasize its importance not only in localization, but also in demonstrating the existence of a lung abscess in doubtful cases as well. In cases in which the "foul" odor is in question, a bronchoscopist of experience is almost always able to decide, after the bronchoscope has been inserted, whether it is the specific odor of anaerobic putridity or otherwise.

PLEURAL COMPLICATIONS

For the sake of completeness the most important features of this subject are discussed here; a more thorough discussion has already been published separately.[9] It is hardly necessary to reemphasize the importance of the pleural reaction that occurs in lung abscess, not only as its usual accompaniment, but sometimes as the dominant feature in the disease. In the majority of cases pleural adhesions are formed very quickly and, as a rule, are sufficient to protect the free pleural cavity from infection. They are the sequels to the localized fibrinous pleuritis which develops over the site of the abscess by virtue of its characteristic superficial situation.

In addition to the above, three main types of suppurative complication may involve the pleura. The first of these develops without warning as an acute hemorrhagic pleurisy which quickly becomes purulent. In this type of putrid empyema the patient has very little or no cough or sputum and is never aware of a foul odor. Aspiration of the empyema yields foul-smelling pus. At operation the abscess of the lung is found to be small and very superficial, and to have emptied its putrid contents into the pleural cavity instead of into a bronchus. Despite severe symptoms and desperate early course, these patients often make an excellent recovery.

A second type of putrid pleurisy is that in which rupture of the abscess takes place after the usual symptoms of lung abscess have been established. This type is fairly readily recognized if the possibility of its existence is kept in mind, as discussed previously. In this type of case, the abscess cavity already communicates with a bronchus so that

air as well as pus escapes into the pleura (putrid pyopneumothorax). In many cases the empyema is of the encapsulated variety.

The third type of pleural complication is, in effect, a sequel to either of the two just described. This is the chronic putrid pyopneumothorax with pleuropulmonobronchial fistula. The latter is rather easy to diagnose in retrospect from the patient's history. The striking event in the past illness is the history of empyema, in some cases proved to have been putrid, which has been inadequately drained if at all. An antecedent history of lung abscess may also be obtained. The course of such patients is one of continued illness until the time of a sudden expectoration of a huge amount of intensely foul pus; this usually affords a period of great relief, but eventually the original symptoms return. In such cases the empyema cavity is the reservoir for accumulation of foul secretion which is periodically expelled through a bronchial fistula into the lung and coughed up in large quantities. As would be expected, marked secondary changes are always present in the affected lung, viz., widespread inflammation and fibrosis, bronchiectasis, and multiple putrid abscesses. Among the rarer pleural complications may be mentioned interlobar empyema and empyema necessitatis with extension either through the chest wall to the skin or into the mediastinum, pericardial cavity, or paravertebral soft tissues.

SUMMARY AND CONCLUSIONS

1. Careful study of clinical symptomatology is often useful in the differential diagnosis of obscure pulmonary diseases. The diagnosis of putrid lung abscess can usually be made from the history alone.

2. Putrid lung abscess is produced by infection of the lung with anaerobic microorganisms aspirated from the mouth of pharynx. Surgical procedures in these regions and blunting of the natural protective reflexes of the pharynx and larynx are the chief contributing causes of inoculation.

3. In nearly half the cases no causative factors are known. The possible importance of badly diseased teeth and gums is suggested.

4. The onset in typical cases of lung abscess is like that of acute bronchitis or grip, or a mild lobar pneumonia. Pleuritic pain is almost always present. The clinical course of the primary stage is marked by mild character and exceedingly low mortality.

5. The gangrenous nature of the underlying disease is revealed, only after an interval of time, by the appearance of blood in the sputum and the large amount of foul-smelling pus. In most cases this occurs by the end of the second week.

6. The typical roentgenogram reveals a single pneumonic shadow with a central spherical cavity. The variation in width and density of the infiltration about the cavity is important as a guide to the behavior of the lesion.

7. Spontaneous remission of some degree is usual during the primary stage of the disease. This is a crucial period in the course of the disease. Complete cure at this stage takes place spontaneously in about 20 per cent of cases, usually cases of mild character. The only safe criterion of cure is disappearance of all symptoms and of the roentgenographic shadow completely.

8. Relapse into a chronic progressive form of the disease is the rule in the majority of cases of lung abscess untreated during the primary stage. The chronic phase is usually definitely established in three to four months. If untreated, this type of case has an average life expectancy of only three years. The bad prognosis of untreated or improperly treated lung abscess ranks with that of cancer.

9. Fibrosis and bronchectasis are the usual accompaniments of chronic lung abscess and may overshadow and obscure the original condition.

10. Individual differences in symptoms of onset, pain, fetor, bleeding, clubbing, and degree of toxemia are of much importance in the evaluation of the type of case, its prognosis and indication for treatment, and in the diagnosis of complications.

11. It is incorrect to speak of postpneumonic or metapneumonic lung abscess; such cases are to be regarded as primary lung abscess with hyperacute onset.

12. The onset and early course of putrid lung abscess in young children is often atypical or unrecognized.

13. The foul odor of putrid abscess is distinguished from other foul odors by its pungent or acrid quality. Variations in intensity of the odor may have considerable importance in an individual case. It is never completely absent unless the gangrenous focus ruptures very early into the pleural cavity before spontaneous bronchial drainage occurs.

14. Putrid superinfection of other diseases of the lung is rare. It is usually easy to differentiate from primary putrid lung abscess by means of the history.

15. The location of chest pain generally indicates the site at which the abscess is most superficial and therefore most accessible to the surgeon. The densest adhesions are found here.

16. Bleeding is more common in lung abscess than in tuberculosis or carcinoma. It may very occasionally be fatal.

17. Clubbing of digits appears earlier in lung abscess than in any other pulmonary disease and disappears only when all infection has subsided.

18. Physical signs of acute lung abscess are of secondary importance because of their indefiniteness. When marked signs exist in an early case, one should suspect either a complication or an incorrect diagnosis.

19. The most useful objective guide to the course of the disease is the roentgenogram. For localization a lateral film is advisable as well as posteroanterior films.

20. The most important intrathoracic complication commonly encountered is putrid empyema or pyopneumothorax resulting from perforation of the abscess into the pleura. This may persist in chronic form.

The authors express their appreciation to Dr. Harold Neuhof and Dr. Harry Wessler for their constructive criticism and numerous suggestions.

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940 PARK AVENUE

CLINICALLY SIGNIFICANT IRREGULARITIES OF THE DIAPHRAGM*

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RECOGNITION of the dense roentgen ray shadow or image cast by a normal diaphragm is roentgenologically made possible by the contrast producing air content of the lungs; in addition on the left side, by the gas containing stomach and colon. This roentgen ray shadow or image is usually described in the following terms: shape that of a dome, contour regular uniform, symmetrical, right diaphragm slightly higher than left with broader lung field above it (because of the liver), cardiophrenic and costophrenic angles relatively acute. Many are wont to consider any deviation from the above, any irregularity in the contour of the diaphragm as being due to some supra- or infradiaphragmatic inflammatory process, or, in other words, label such an irregularity as a true adhesion. Such a conclusion may lead one astray frequently. The presence of a true inflammatory irregularity of the diaphragm may lend weight sufficient to clinch an otherwise uncertain diagnosis or to preclude or suggest the use of certain therapeutic measures.

In this brief presentation it is my purpose to draw attention to the following: (1) that not every irregularity in the contour of the diaphragm need always be considered as evidence of a true adhesion or of an organic lesion; (2) that the presence of a pulmonary lesion does not permit one to conclude that an associated irregularity in the contour of the diaphragm is a pathologic one. I shall consider in particular so-called visual insertions of the diaphragm and double contours.

It is gradually being appreciated that an adequate examination of the chest consists not only of the ordinary physical examination but also of a complete roentgen ray examination. From this standpoint we have now come to learn too that only by attempting a pneumothorax is it possible to tell whether it will be feasible in the given case. The same holds true for the cutting of pleural adhesions. Those who have had experience with intrathoracic hernia know that the first question to be answered, when the physical examination suggests an elevated diaphragm or displacement of the heart, is whether or not both diaphragms are present and intact. This question can be satisfactorily answered on most occasions by fluoroscopically observing the patient after he has taken a drink of barium. This simple procedure in addition gives information concerning the presence or absence of the so-called normal milking action

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of the diaphragmatic pinchcock. A proper examination of the diaphragm also demands that the patient be examined in various positions. Thus only with the patient in the various oblique and lateral positions is it possible to study that portion of the diaphragm which is hidden behind the heart shadow. The presence of a hiatus or abnormal recess in the diaphragm can be recognized best by examining the patient in this way.

Visual insertions or individualization of the costal components of the diaphragm according to Weltz, Glauner and others occur in singers and individuals suffering from asthma, emphysema, chronic bronchitis and fibrotic lesions of the lung. The condition may apparently be produced experimentally by cutting both phrenic nerves. I have also observed it and double contours of the diaphragm with phrenic neuritis. It has also been described in individuals suffering from strychnine poisoning, and has been recognized too in the presence of an unstable mediastinum, tension pneumothorax and with scoliosis. That it may be seen quite independent of any demonstrable organic lesion in the body is appreciated.

A variety of theories has been offered in explanation of the condition. Among them may be numbered a deep inspiratory position of the thorax, a change in the form of the thoracic cage, a straining or tension of the diaphragm, variations in intrathoracic pressure, hypertrophy of certain muscle bundles of the diaphragm and furrows in the liver. It is not unlikely that more than one mechanism plays a part in the production of the condition. Weltz and Glauner, upon the basis of good evidence, are firmly convinced that the concave portion of the deformity represents rib attachments of the diaphragm. They have demonstrated that the convex contours of the diaphragm appear to be responsible for liver furrows in some cases. There seems to be some association between duplications of the diaphragm, on the one hand, and furrows or ridges in the liver, on the other hand. The latter may be observed in individuals suffering from chronic bronchitis, emphysema, asthma, and scoliosis but may exist independently.

During the past year and a half I have observed visual insertions on more than fifty occasions. In one-half the cases the condition was physiologic. In the majority of the other cases it was observed in asthmatic persons and in individuals suffering from emphysema or scoliosis.

On three occasions it was associated with pain referable to the right shoulder. In every instance the distribution of pain was that found in the presence of a phrenic neuritis.

On several occasions it was found that the diagnosis of pleurisy with adhesions had been made solely upon the basis of what were undoubtedly physiologic visual insertions. On other occasions the presence of a

pneumothorax had immediately led to the conclusion that the steplike irregularities present (visual insertions) represented fluid.

The roentgenologic appearance of visual insertions of the diaphragm can best be appreciated by consulting Fig. 1.

How then can one differentiate between visual insertions of the diaphragm and true adhesions? The answer, I believe, lies chiefly in thinking of the possibility of the condition. When the lung is pushed away from the diaphragm, that is, when a pneumothorax is present, then it is a relatively simple matter. Under such circumstances as already mentioned it has not been uncommon for some to consider the steplike diaphragmatic extensions to the ribs as collections of fluid. The relationship of the irregularities to the ribs then is also an important consideration. Certainly visual insertions are more frequently multiple. The fact that visual insertions of the diaphragm can usually be seen

Fig. 1.—*A*, Hypertrophied costal components of the right diaphragm, (indicated by arrows, after Weltz and Glauner). Through the courtesy of Dr. David Seecof, director of laboratories, Jewish General Hospital, I recently obtained a similar specimen. This illustration clearly indicates the type of roentgenologic picture to be expected in the presence of such a finding. *B*, "An oblique view showing the relationship of each of the steplike diaphragmatic extensions to the individual ribs." (After Fleischner, taken from Singer and Boikan.)

only during deep inspiration also serves to distinguish them from adhesions.

To avoid confusion I have purposely delayed referring to Matson's observations until this point. Matson in 1922 drew attention to the fact that "the assumption that tenting of the diaphragm always diagnosticates pleuro-diaphragmatic adhesions is unjustifiable." He considered tenting of the diaphragm as a purely physical phenomenon brought about by two factors in the following manner: (a) "During inspiration the diminished elasticity or rigidity of the lower bronchial branches prevents descent of that area of the lung base in immediate relationship to the bronchus involved; thus a dimple effect is produced on the surface of the lung. (b) The diaphragm being molded to the base of the lung by a

negative intrathoracic pressure is held by suction in the dimple referred to above producing a tented appearance (pseudo-adhesion)." Matson also pointed out that such pseudoadhesions may be found during the course of acute and chronic respiratory infections, that they may disappear, but that they can also be converted into true adhesions should the pleura contiguous to the diaphragm become involved by an inflammatory process. Certainly so-called tenting, in which a triangular shadow with the base toward the diaphragm is observed best and frequently not at all save during deep inspiration, can be but a physiologic variation in the contour of the diaphragm. Such may be the case even when this variation is single, although Singer and Boikan would have us believe that they can be considered as physiologic only when they are multiple.

Double contours of the right diaphragm are frequently encountered in individuals suffering from bronchitis, asthma and emphysema. It may also be observed in the presence of tracheal stenosis, bronchial stenosis, and infiltrative processes of the lung. This would suggest that variations in intrapleural pressure may play a part in their production. In some necropsy cases furrows in the liver have been demonstrated. I have also observed double contours of the right diaphragm following phrenicectomy. It is thus possible that in the given case unequal atrophy of the musculature of the diaphragm had taken place. The possibility that only certain muscle bundles of the diaphragm had had their innervation interrupted must also be considered under such circumstances.

The roentgenologic appearance of the double contours of the diaphragm can be appreciated best by consulting Fig. 2. The medial arc is usually higher and more convex than the lateral one. It, too, is observed best when the patient is made to take a deep inspiration. While visual insertions have been observed below the diaphragm in the presence of a pneumoperitoneum, to my knowledge such has not been the case with double contours. There is no definite evidence that increased intra-abdominal pressure plays a part in the production of either.

Fluoroscopically double contours may be observed on some occasions during expiration but are observed best after deep and rapid inspirations. The condition at first glance suggests that one part of the diaphragm moves better than the other or that one part moves and the other does not. They can be made to disappear by having the patient bend forward.

Eisler and Thomas (cited by Singer and Boikan) offer the following explanation for the medial anterior bulge. "These authors ascribe the phenomenon to the varying length, strength and position of the component muscle bundles of the diaphragm. These may be divided into a powerful posterolateral group and a much weaker anteromedial group. The former is composed of the posterior lumbar fibers forming the crura

diaphragmatici and the lateral costal fibers from the ninth rib down. This group inserts into the posterolateral part of the right wing of the central tendon. The contraction of these fibers pulls the posterolateral part downward very forcibly. In contrast the anteromedial part of the diaphragm is depressed with relatively little force by the short fibers arising from the sternum, xiphoid, and the seventh and eighth ribs."

According to Assmann the absence of a large perforating vein together with the presence of the heart on the left side accounts for the absence of similar changes in the left diaphragm.

Singer and Boikan state that the actual groove separating the anteromedial from the posterolateral cupola is formed by the straddling action of the strong fibers from the crus medialis joining the fibers originating from the ninth rib. They are of the opinion too that the smaller medial

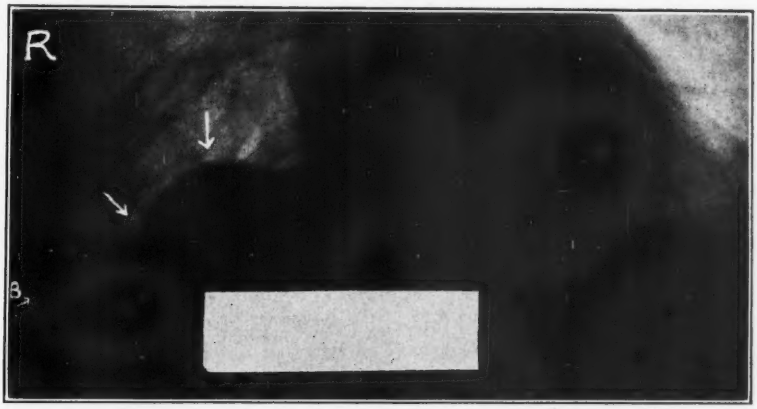

Fig. 2.—Double contour of right diaphragm. *R*, Right; *B*, breast shadow. Arrows indicate double contour; save for increased hilus detail in the right lung, the findings in this case were essentially negative. (Patient of D. Barza and D. L. Mendel.)

bulge is formed by the short anteromedial group of muscle fibers mentioned previously, and posteriorly by the right wing of the central tendon.

What has been said about recognizing visual insertions also holds true for double contours of the diaphragm. One must think of the possibility of the condition for it is of frequent occurrence. Repeatedly similar roentgen ray pictures of the right diaphragm suggestive of a double contour of the diaphragm should be considered as physiologic until proved otherwise. Only when repeated roentgen ray films and fluoroscopic examinations show a varying finding and then only in the presence of a positive clinical history substantiated by positive signs and symptoms should such an irregularity of the right diaphragm be considered as suggestive of a supra- or infradiaphragmatic process. As already noted, double contours of the right diaphragm are best observed

during inspiration and can be made to disappear by having the patient bend forward.

On some occasions in order to establish the diagnosis I have found it necessary to perform not only a lipiodol injection of the corresponding lung but also a pneumoperitoneum.

I should like to refer to the so-called saucer deformity of the diaphragm. When associated with intraabdominal suppuration it may suggest the appearance of a subphrenic abscess. This deformity cannot be attributed to dual motor innervation of the diaphragm as suggested by Middleton, for the motor innervation of the diaphragm is solely through the phrenic nerve.

I have observed several such deformities of the diaphragm in one case in association with a subphrenic abscess which was drained. A considerable time after the abscess had healed the diaphragm failed to return to its normal level nor did it assume its normal contour. A secondary pleural effusion had been present in this case and had been coughed up through a bronchopleural fistula. The fluid was replaced by air which had spontaneously escaped into the cavity. Some of this air rested over the diaphragm after the bronchopleural fistula had healed. Some few true adhesions persisted. A further quantity of air was injected into the pleural cavity. It caused the diaphragm to return to its normal position but not entirely to its normal shape. Since this patient has been well for almost two years, I am inclined to believe that it is not unlikely that one was dealing with a diaphragm which originally exhibited a double contour. In this instance the persistent irregularity for some time suggested that the subphrenic abscess had not been completed and adequately drained.

Occasionally an individual presents himself with a neglected or what one usually calls a chronic empyema. There is no history of a previous true pneumonic process. In such cases the withdrawal of pus should always be followed by replacement with a small quantity of air if a definite fluid level is not already present. Films should then be taken in such a way as to visualize the diaphragm. If it then appears as an elevated straight line, with the elevation and inclination most marked at the costal margin, then until proved otherwise such a finding has to be considered as being due to an infradiaphragmatic process. The possibility of a subphrenic abscess must be considered in every such case.

It should also be noted that an interlobar effusion may rest over the diaphragm in such a way as to suggest a double diaphragm (one diaphragm upon the other) but not a double contour in the strict sense of the term.

SUMMARY

In this presentation attention has been drawn to the fact that not every irregularity in the contour of the diaphragm need always be con-

sidered as evidence of a true adhesion or of an organic lesion. It has also been pointed out that the presence of a pulmonary lesion does not permit one always to conclude immediately that an associated irregularity in the contour of the diaphragm is a pathologic one. Particular reference has been made to so-called visual insertions of the diaphragm and to double contours of the right diaphragm. The frequency with which these irregularities in the contour of the diaphragm are met makes them of more than academic interest, particularly since many of them are suggestive of or are observed in association with organic lesions.

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POSTPNEUMONIC ATELECTASIS AS A POSSIBLE CAUSE OF CHRONIC EMPYEMA*

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IN A PRIOR article[1] it was stated, "Acute empyema does not of necessity go on to chronicity. . . . The factors that predispose to chronicity are threefold: Infection with such organisms as the tubercle bacillus, that give rise to chronic inflammatory processes; low resistance on the part of the patient, due to syphilis, secondary anemia, constitutional disease, or similar cause; and illogical initial care. . . . The immediate causes of chronicity are: Infection by tubercle bacillus or similar organism; faulty mechanics of the drainage system; foreign bodies within the cavity or sinus; osteomyelitis of the ribs adjacent to the drainage wound; pleuro-pulmonary communications and residual foci of infection in the walls of the cavity." It is desired at this time to enumerate a fourth predisposing factor, namely, postpneumonic lobar atelectasis.

In a series of 189 acute empyemas of pyogenic origin, there was recognized in four instances a coincident lobar atelectasis. It is quite possible that the condition may have existed, unrecognized, in other cases. One patient died. In the other three cases the clinical course was unusually stormy, convalescence was delayed, and while fortunately none of these cases became chronic, there was in every one of them a definite threat of chronicity, such as is not observed in the usual case.

One of the four cases has previously been briefly reported before the Pennsylvania State Medical Society;[2] the second was presented before the New York Society for Thoracic Surgery, in March, 1933, but the record has never been published; the other two have never previously been reported.

CASE 1.—EFB No. 612, J. S., male, German, aged seventy years, retired merchant, was first seen Jan. 7, 1928, at which time he was suffering from a lobar pneumonia involving the left lower lobe. Two days later the pneumonia terminated by crisis, but was shortly followed by an acute postpneumonic empyema on the same side. Pneumococcus was recovered in pure culture from the exudate. Following a series of aspirations, open drainage by resection of a portion of the ninth rib was instituted on Jan. 27, 1928. Convalescence was not smooth, the wound was sluggish, the size of the empyema cavity did not progressively diminish, temperature and pulse remained irregular, and the lung, as seen in the depths of the wound, appeared decidedly darker than normal. The roentgenogram showed a persistent shadow in the region of the left lower lobe and a well-drained circumscribed empyema cavity. Except for local conditions in the left lower lobe and pleural cavity, there was no evidence of disease. Malignancy was suspected. Therefore, on Feb. 11,

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1928, the lower lobe was explored through the empyema wound. Neither malignancy nor abscesses were found. A specimen of lung tissue showed chronic fibrosis and polymorphonuclear infiltration. On Feb. 16, 1928, the bronchoscopic examination showed narrowing of the airways to the left lower lobe, longitudinal streaking mucosa, but a complete absence of obstructing secretions. Almost immediately after this examination there was improvement in the patient's condition, temperature subsided to normal, the wound took on a healthy appearance, the cavity rapidly diminished in size, and a month later he was discharged from the hospital with only a superficial granulating area to mark the site of this operative wound.

In November, 1928, there was a recurrence of superficial suppuration in the wound. Another small superficial abscess appeared in the scar in September, 1929. There was no evidence of any involvement of deep structures. Neither of these recurrent abscesses persisted for more than two weeks.

In September, 1933, five and a half years after the original illness, the patient was carefully examined. There had been no further recurrence of suppuration. Health had been good. The patient was still vigorous at the age of seventy-five years. The physical examination of the chest showed such slight changes as might be found in the neighborhood of any empyema scar. Fluoroscopy showed clear pulmonic fields and good motion of the diaphragm on the affected side. Supraglottic pneumonography with iodized oil, 40 per cent, showed no residual atelectasis and no apparent evidence of bronchiectasis.

In March, 1934, six months after the introduction of iodized oil, there was still some oil retention in the lower airways on both sides and some puddling suggestive of slight bronchiectatic changes. The general health of the patient, however, was good.

CASE 2.—EFB No. 891, N. G., male, white, aged seven years, schoolboy. This patient developed a right lobar pneumonia in April, 1932. He was treated at home until his attending physician recognized the presence of a postpneumonic empyema on the right side. He was then transferred to the Tioga County General Hospital for definitive treatment. Aspiration only was carried out until the centrifuged pus showed more than 40 per cent of sediment. Thoracotomy with resection of a portion of rib was done on April 30, 1932, and the cavity irrigated with Dakin's solution. The postoperative progress was not satisfactory. A secondary thoracotomy with resection of the portion of a lower rib was later performed. Blow bottles and breathing exercises were employed. The empyema cavity, however, did not diminish in size, and the temperature remained irregular. The roentgenograms showed an atelectatic lower lobe. On July 22, 1932, bronchoscopic examination was carried out without finding any bronchial obstruction. For some cause, however, the size of the cavity soon began to decrease and within two weeks had become practically obliterated. The child was discharged, healed, in early August.

In February, 1933, the child was reexamined. There had been no recurrence. The wound was well healed; the right side of the chest was limited in size and excursion; the excursion of the right diaphragm was estimated to be 75 per cent as great as that of the left side. There was no fluoroscopic evidence of residual atelectasis and no clinical suggestion of bronchiectasis.

In March, 1934, the child was again examined and the chest found in satisfactory condition, although there was some limitation of motion on the right side. Supraglottic pneumonography with iodized oil showed a complete reexpansion of the right lower lobe, no residual atelectasis whatever, and no evidence of any bronchiectatic changes.

CASE 3.—EFB No. 926, F. MacD. Jr., white, aged ten and one-half years, schoolboy. This patient was taken sick with chills, nausea and vomiting on April 25, 1933. Five days later he was admitted to the Arnot-Ogden Memorial Hospital with

a diagnosis of right upper lobe lobar pneumonia. It is of interest to note that the roentgen ray film on admission showed no pneumonic involvement of the right lower or middle lobes (Fig. 1). The temperature fell by lysis, reaching the low point of 99.2 on May 10, 1933, with a subsequent prompt rise to 102.6. At that time roentgen examination showed a dense shadow occupying the lower half of right chest (Fig. 2). Thoracic consultation was requested; fluid was found in the right chest, and aspiration on May 10, 1933, yielded 135 c.c. of thin, watery pus, from which both streptococcus and pneumococcus were recovered. A second aspiration was performed two days later and thick pus obtained. On May 16, 1933, primary thoracotomy with resection of a portion of the ninth rib was performed, but the

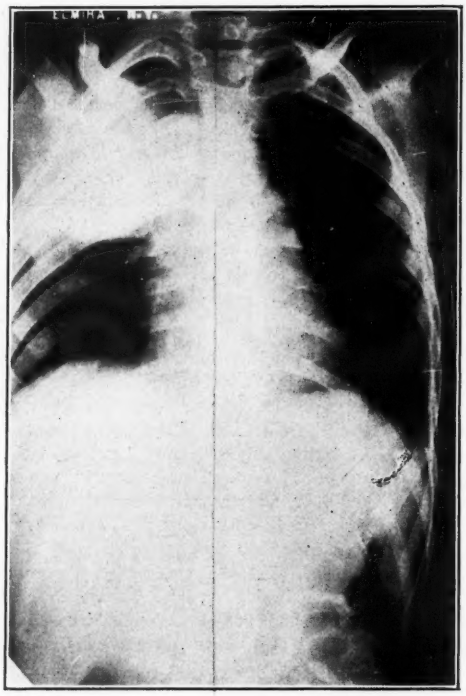

Fig. 1.—Case 3. Roentgen appearance of chest at time of admission to hospital, showing pneumonic involvement of right upper lobe.

wound was allowed to remain temporarily occluded for an additional forty-eight hours, at the end of which time the temporary sutures were removed and the wound was left wide open. There was no respiratory embarrassment, and routine irrigation with Dakin's solution was instituted. Convalescence was protracted. It was marked by a secondary anemia, the red cells falling as low as 2,800,000. One transfusion was carried out. The temperature ranged irregularly between 99.4° and 102.4°. At no time was the capacity of the cavity unduly large, and by June 20 it was obliterated. Fluoroscopy at that time, however, showed the presence of a right basal paramedian shadow, together with decreased motion of the diaphragm on that side. Bronchoscopic examination on June 21, 1933, showed the airways leading to the right lower lobe to be narrower than normal, decidedly in-

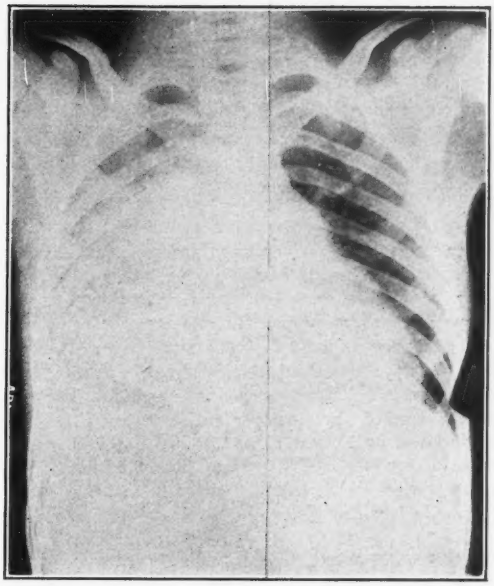

Fig. 2.—Case 3. Roentgen appearance of chest fifteen days after admission, showing empyema at right base and resolution of right upper lobe.

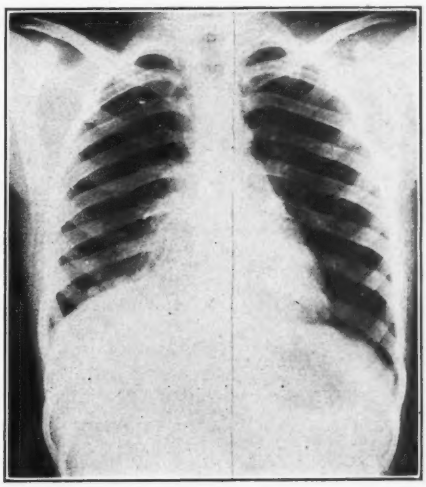

Fig. 3.—Case 3. Roentgen appearance of chest after bronchoscopic introduction of iodized oil, 40 per cent, into right lower lobe bronchi. This film was made forty days after that in Fig. 2. The empyema cavity was practically obliterated, but there remained a right lower lobe postpneumonic atelectasis.

flamed, but free of secretions. Roentgen ray films after bronchoscopic introduction of iodized oil, 40 per cent, clearly demonstrated a residual atelectasis of the right lower lobe, thereby explaining the right paramedian shadow (Fig. 3). On June 25 the wound was entirely healed. By July 1 his weight had increased to 71 pounds. Fluoroscopic examination disclosed the same right paramedian shadow. On August 1, 1933, there had been an additional gain in weight to 75.5 pounds. There was no longer any fluoroscopic evidence of residual atelectasis.

By January, 1934, the weight had increased to 83 pounds. The child was well. There was no fluoroscopic evidence of any residual paramedian shadow, and the recovery of the patient was apparently complete.

CASE 4.—EFB No. 721, A. W., female, white, housewife, aged thirty-two years.

During January, 1929, the patient developed pneumonia involving the right lower lobe. Two and one-half weeks after onset, there was evidence of fluid in the right pleural cavity. Aspiration showed the presence of thin, foul, blood-tinged pus. Pneumococcus, Type III, was recovered. In this case the empyema was evidently secondary to a lung abscess. Closed drainage was instituted and maintained for eleven days. Open drainage was then substituted, and for three weeks the patient showed definite improvement. Then an acute pneumonitis developed on the opposite side, coupled with a marked thyrotoxicosis. The patient's condition rapidly became worse, and she died on February 28, 1929. Autopsy showed an acute pneumonia of both lobes on the left side, 500 c.c. of clear fluid on the left, a practically normal right upper lobe, a healing abscess cavity in the right lower lobe, and an atelectatic right middle lobe.

Of the four cases reported, three patients recovered and one died. The fatal case can be eliminated from further consideration, inasmuch as the atelectasis found at autopsy probably did not modify the clinical course. In the three surviving patients the time interval from definitive operation to obliteration of the empyema cavity was respectively fifty days, ninety-seven days and forty days. These intervals are certainly longer than average. In all three cases there was protracted elevation of temperature. Figures showing the capacity of the various cavities are lacking, but in Case 1 there was no change for a long time. In Case 2 there was a persistent large cavity with no measurable decrease in capacity. In Case 3 there was slow progressive obliteration. In Case 1 the originally involved lobe was the seat of the atelectasis; in Case 2 it is impossible to state accurately which lobe was first involved; in Case 3 it is very definitely apparent, both from the clinical record and from the roentgen examinations, that the atelectatic lobe was not involved in the initial pneumonic process. Two cases occurred in elderly patients, two occurred in children.

References to postpneumonic atelectasis are rare in textbooks and infrequent in the literature. Heuer[3] includes among the causes of chronic empyema "Nonexpansion of the lung following fibrosis and collapse." Such fibrosis might be expected in lobes compressed by exudate or in atelectatic lobes. MacCallum[4] noted a "patchy atelectasis" in autopsy specimens obtained during the influenza pandemic of 1918. Bradford,[5] in 1919, reported lobar atelectasis occurring as a sequel to lobar pneu-

monia. Norris and Landis[6] mentioned atelectasis as a complication of bronchitis and bronchopneumonia. They further stated that pleural exudates may, by compression, produce atelectasis of the adjacent lung. Peppard[7] described the condition at length in 1927 and reported four cases. He concluded that "massive atelectasis occurs as a complication of pneumonia," but did not state the percentage incidence of this complication. In none of his cases was there a coincident empyema. Coryllos and Birnbaum[8] in 1929 considered lobar pneumonia "as an 'infectious (generally pneumococcic) lobar atelectasis of the lung.'" Lord[9] has found "a recognizable atelectasis occurring in a small proportion of patients with pneumonia" and has also observed "the combination of bronchopulmonary infection, bronchostenosis and empyema—a number of times." Many authors have referred to pulmonary fibrosis and bronchiectasis as late sequellae to pneumonia.

The diagnostic signs of lobar atelectasis, pure and simple, are well known and have been repeatedly described at length. It does not necessarily follow, however, that the same physical signs would be found in atelectasis occurring coincidently with empyema. Prior to drainage of the empyema the presence of the exudate will mask the atelectasis and control the physical signs and roentgen ray findings. The volume of exudate will effectually prevent the characteristic displacement of the heart and mediastinum toward the affected side, and may even be great enough to displace the heart and mediastinum toward the unaffected side. After drainage of the empyema physical examination will be hampered by the presence of the wound, and the physical signs will still be modified by the existence of the empyema cavity within the affected hemithorax. In the cases here reported there was no displacement of the heart and mediastinum toward the involved side. All these patients were subjected, ultimately, to open drainage. Possibly if closed drainage and negative pressure had been maintained throughout the entire course, there might have been such a displacement.

The most reliable diagnostic procedures are roentgen ray examination, pneumonography with iodized oil, 40 per cent, and bronchoscopic examination. The roentgen ray will show increased density and decreased volume of the affected lobe. Pneumonography will show the airways within the atelectatic lobe to be either occluded or definitely narrowed and lying abnormally close together. Bronchoscopic examination will show grossly narrowed or even occluded airways leading into the affected lobe. The diagnosis, in the four cases here reported, was made at autopsy in one case, was suggested by the basal paramedian roentgen shadow and confirmed by bronchoscopic findings and pneumonography in another case, and was established by bronchoscopic examination in the remaining cases.

In two cases here reported (Cases 1 and 2) the atelectasis was not only found by endobronchial examination, but was also dramatically relieved

at the same time. The relief was so prompt and striking that it deserves special comment. In neither case was there gross evidence of occluding secretions within the airways. The bronchi were narrowed but apparently dry. In each case the smaller branch bronchi were explored with small curved aspirating tubes. In each case reexpansion of the lobe was rapid from that very moment. This phenomenon is best explained by assuming that the compressed bronchioli or alveoli of the atelectatic lobe are still filled with viscid secretions which have resisted ordinary respiratory effects at removal. Endobronchial manipulation effects the reopening of at least some of the bronchioli, and air gains access to at least some alveoli. This permits the collateral respiration described by Van Allen and Jung[10] in 1931 to reestablish rapidly the patency of the entire lobe.

As already mentioned, the most effective therapeutic measure is endobronchial manipulation. Other possible measures are forced hyperventilation with carbon dioxide, blow bottles, atropine to inhibit spasm of the bronchopulmonary smooth muscle, and possibly adrenalin as a stimulant to sympathetic activity.

CONCLUSIONS

1. Postpneumonic atelectasis and postpneumonic empyema may occur coincidently. This relation has been observed in slightly more than 2 per cent of a small series of postpneumonic empyemata.

2. Postpneumonic atelectasis, when occurring coincidently with postpneumonic empyema, modifies the course of the empyema, delays ultimate recovery and predisposes to chronicity.

3. The most reliable diagnostic procedures are roentgen ray examination, pneumonography with iodized oil, 40 per cent, and bronchoscopy.

4. The most effective therapeutic measure is reestablishment of collateral respiration within the atelectatic lobe by endobronchial aspiration.

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EXPERIMENTAL TOTAL PNEUMONECTOMY*

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ALTHOUGH thoracic surgery has made great progress in recent years, it is not a new field. Records of thoracotomy for empyema along with recognition and drainage of pulmonary abscess can be traced back to the writings of Hippocrates in 400 B.C. The subject seems to have been either avoided or neglected until the end of the sixteenth century when Schenk in 1584 called attention to this important branch of surgery. John Bell in 1807, far in advance of his time, described clearly and accurately the symptoms, signs, and prognosis of wounds of the pleura, lung, and heart. In 1878, Grinnell[1]† reported the successful removal of a considerable portion of the lower left lung, which had been pulled through the chest wound in the futile attempts on the part of the patient, an Indian, to extract a barbed arrow. Although considerable suppuration followed, the patient finally recovered.

The earliest experimental work on resection of the lung was reported by Gluck[2] in 1881 when he removed the entire lung on one side from 6 dogs and 14 rabbits. With the exception of 2 rabbits, all died in from seven to ten days. Marcus'[3] and Block's[4] experiments performed in the same year were equally discouraging. Biondi[5] (1882) reported his results, which were the best obtained in these early investigations. Of 11 pneumonectomies, only 4 patients died of septic pleuritis. Zakharevitch's[6] (1890) results equalled Biondi's. In 1891, Willard[7] performed

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†"Some Caddoe boys were practicing with bows and arrows, when one about eight years of age received a shot in the lower portion of the thorax, between the fifth and sixth ribs, just to the left of the median line.

"The barbed arrowhead entered deeply into the lung.

"The boy immediately grasped the shaft and drew out the arrow—the head carrying with it, through the orifice, quite a large portion of the lung, not, however, severing it from the remainder of the organ. The family lived some fifteen miles from my office; and the 'medicine man' near at hand who was summoned, was unable by his blowing, howlings, or enchantments to make the lung replace itself or to remove it. He decided that the part thus drawn out was 'fat,' and that was as far as he could go.

"I did not see the case until twenty-four hours after the injury was received. When seen the exposed lung was congested, so that it resembled the liver or spleen, more than lung tissue. The weather was exceedingly warm and the odor was already becoming offensive.

"Chloroform was at once administered, and after placing a ligature around the part close to the wound, I removed the portion of the lung exposed to view. Then washing the portion below the ligature which still tended to protrude—with a solution of per chloride of iron—I returned it within the small opening made by the arrow, and applied sutures.

"The portion of lung thus removed was 4½ inches in length and 2¾ inches in breadth at the widest part.

"Some degree of suppuration took place a few days after, and two weeks after the operation the ligature came away with a quantity of pus. Since that time the boy has steadily improved. He has now quite recovered, and is resuming his usual sports."

a successful pneumonectomy in a dog by suturing the stump which he had ligated en masse into the wound. Murphy[8] (1898) lost 8 out of 9 dogs with septic pleuritis. In 1906, Green[9] resected parts of lungs in 14 dogs, losing 7 of pleuritis. The following year Tiegel and Mitt[10] called attention to the fact that ligation en masse was a primitive method. They advised loose mass ligation with silk about the hilum, followed by peripheral ligation of the vessels. Following resection, the bronchial stump was cauterized with heat and ligated distal to the first ligature. His method also was followed by the complications of hemorrhage and bronchial fistula. Frederich[11] (1908) advised separate ligation of vessels. Following resection of the lung he removed the mucous membrane, after which the bronchus was ligated to bring the raw surfaces into apposition. Danielson[12] (1908) concluded that it was possible to close the main bronchus with silk. In the same year Halsted[13] reported 21 thoracotomies with only one subsequent infection in the pleura. In this respect his results were quite different from those of earlier investigators. He advised bisecting the bronchus and removing the mucous membrane, followed by suturing of the flattened halves of the bronchus with through-and-through silk sutures together with a series of sutures placed around the edges. Willy Meyer[14] (1908) described his inversion method in which the bronchial mucosa was undisturbed and the closure of the bronchial lumen accomplished at the expense of the peribronchial tissues. In 21 dogs he crushed the main bronchus and ligated it with a transfixion suture of medium silk. Of these 21 dogs he lost 4, a mortality of 19 per cent. Robinson and Sauerbruch[15] removed one lung from 38 animals with only 4 recoveries, using ligation en masse. Schlesinger's[16] results (1911) were similarly poor. Quinby and Morse[17] concluded that the method as described by Meyer seemed the most accurate and successful after treating each bronchus separately in 38 dogs with only 8 deaths.

Garré[18] (1912) advocated the sewing of the lung over the opened bronchus, after having placed a ligature about the bronchus. Henschen[19] (1914) suggested the introduction of a plug of fascia into the bronchus and closing the stump over it. Giertz[20] undertook the problem but modified it by using silk mattress sutures to close the bronchial stump; following which he covered it with small fascial transplants sutured with silk to the peribronchial tissues. Of 10 dogs operated upon, he lost 2 of tension pneumothorax. Kawamura[21] (1914) modified the method of Tiegel by using a proximal bronchial ligature. Only 2 bronchial fistulas resulted in the 23 pneumonectomies performed on dogs.

Heuer and Dunn[22] (1920) employed all the previous methods described along with several new ones with 13 recoveries in 23 pneumonectomies. Feiermann[23] (1925) after reviewing the various technics of treatment of the bronchial stump considers the method as described by Willy Meyer the best, but suggests the use of a reverse purse-string suture

to the peribronchial tissues to give the stump a greater solidity. Bettman and his coworkers[24] (1928) reported an excessively high mortality in both lobectomies and pneumonectomies, losing 25 out of 27 dogs, and concluded that the closure of the bronchus was difficult on account of the "inherent inertness" of the bronchial wall tissues. Joannides[25] (1928) found that primary bronchi are hard to invert and that the results are bad. He consequently advised amputation of the lobe to be done through the lung tissue. All his dogs, however, showed evidences of pleural irritation with greater or lesser amount of exudation. Furthermore, at autopsy the site of suture was found always to be adherent to the thoracic wall. Schleuter and Weidlein[26] (1928), believing the invagination method as proposed by Willy Meyer to be the most ideal procedure for lobectomy, modified it by cutting the primary or secondary bronchus between ligatures, then inverting it with a No. 1 chromic catgut purse-string suture and in the case of a primary bronchus reinforcing it by three mattress sutures of silk. Following this, a final purse-string suture was placed to cover the mattress sutures with the looser peribronchial tissues. In 13 lobectomies on dogs in which this method was employed, 1 died of empyema, 1 of bronchopneumonia, 1 of distemper, and 1 of a cause unknown. In 4 pneumonectomies performed by treating each bronchus separately, one died of empyema on the fourth day. Nisson[27, 28] (1931) in the attempt to minimize the dangers of the complications following ligature en masse devised a two-stage procedure; surrounding the lobe to be excised with a coarse silk mesh bag to obliterate the pleural cavity, followed by resection and ligature en masse two to four weeks later. Adams[29] (1931), in order to reduce the dangers of bronchial fistula, applied 35 per cent silver nitrate to the bronchial lumen to produce bronchial stenosis prior to resection of the lung. Bryce[30] (1934) suggests a three-stage procedure: (1) the production of adhesions by a large mesh bag (Nisson); (2) the ligation of the pedicle of the lung followed by (3) the removal of the necrotic lobes. Sixty-six and six-tenths per cent of his animals (cats) survived this procedure.

Review of the literature not only of experimental lobectomy and pneumonectomy but also of the clinical fields (Hale,[31] Grinnell,[1] Duvon,[32] Morrison,[33] Robinson,[34-36] Lilienthal,[37-39] Sauerbruch and Robinson,[40] Graham,[41, 42] Hinz,[43] Churchill,[44] Alexander,[45] Edwards,[46] Brunn,[47, 48] Shenstone and Janes,[49] Eloesser,[50] Rienhoff,[51] Roberts and Nelson,[52] Heuer[53]) reveals that while hemostasis is the least troublesome factor, the obtaining of a completely air-tight bronchial stump is the most difficult as well as most important phase of the problem. The high incidence of bronchial fistula along with the high incidence of pleural infection are to be ascribed essentially to the inherent difficulties in the care of the bronchial stump. It is the *bête noire* of thoracic surgery. Sauerbruch very sagaciously summarized the problem, "Bei Lungen-

amputation muss der grösste Wert auf eine zuverlassige Versorgung des Bronchialstumpfes gelegt werden. Sei ist ein technisches problem, das noch nicht vollstandig gelöst ist.''

The inherent difficulties are to be explained on the basis of the singular anatomic construction of the bronchus. The bronchus is composed mainly of cartilage, which is prone to undergo degenerative changes when subjected to trauma. The poor blood supply, derived from the bronchial arteries, increases the inherent tendency of the cartilage to degenerate. In addition, the position of the cartilaginous rings continually tends to separate the opposed bronchial walls. It has been shown by Bettman and his coworkers[24] that the healing of a bronchial stump is brought about by the peribronchial tissues, which are also supplied by the bronchial arteries. Any interference with the meager blood supply of these structures by rough or careless handling of the stump will lead either to a failure of the peribronchial tissues to participate in the process of repair or to necrosis of the tissues with the inevitable development of a bronchial fistula. In analyzing earlier experimental work it will be noted that the stripping from the bronchus of the peribronchial tissues has been an important factor in the failures of many investigators.

The fact, also, that the healing is brought about only by the peribronchial tissues, the blood supply of which is meager, indicates very clearly that the reparative process must needs be slow and that a considerable time must elapse before the closure of the bronchial stump may be regarded as solid. Added to this, the fact that the mucous membrane of the bronchial tree is frequently contaminated with buccal organisms requires that no suture used penetrate into the bronchial lumen. The suture hole will act as a tract along which the bacteria may grow, giving rise to an infection not only of the bronchial stump but also of the pleura.

It would appear, therefore, that the most ideal operative technic for the treatment of the bronchial stump will be that method in which meticulous care is taken not only to prevent infection but also to avoid disturbing the normal continuity of the structures at the hilum, along with the minimizing of trauma to the bronchus.

The technic as described and executed by Willy Meyer meets essentially most of these requirements with the following exceptions: (1) the devitalization of bronchial tissue by means of a crushing clamp; (2) the difficulty encountered in bringing together the edges of the invaginated border without piercing through the wall into the bronchial lumen with the sutures; (3) the necessity for turning in an excess amount of bronchial wall the blood supply to which is interfered with by the acute angulation; (4) the difficulty encountered in attempting to invert a main bronchus of large dimensions by this method.

In the attempt to overcome the above disadvantages certain modifications were made and the following technic was executed in 30 dogs.

Technic.—Preoperative Medication: Morphine ¼ gr. thirty minutes before operation. Anesthesia: Intratracheal ether using an Erlanger positive pressure apparatus with oxygen attachment.

The thorax is shaved on one side from the dorsal spine to sternum and from the second to the twelfth ribs inclusive. The skin is prepared by thorough scrubbing with soap and water followed by benzene, alcohol, and ether. The skin is then painted with iodine and the excess removed with alcohol followed by ether. A long linear incision is made

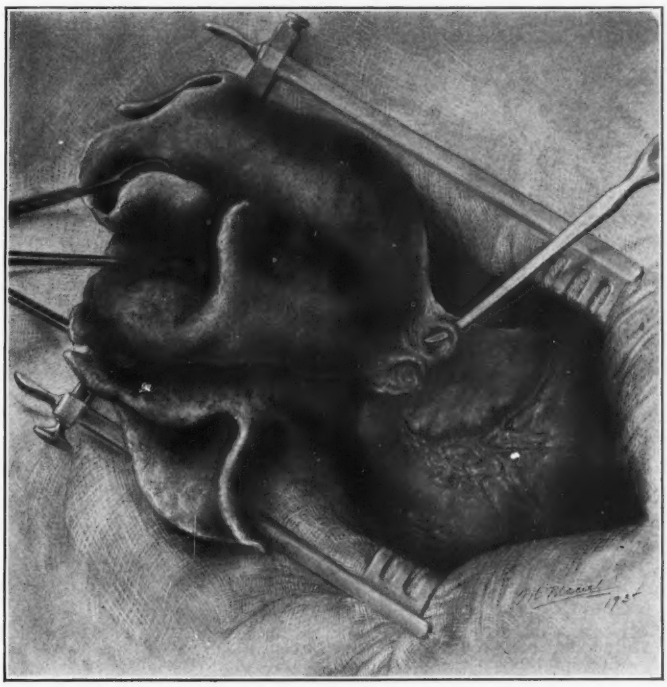

Fig. 1.—The pulmonary ligament has been incised up to the pulmonary vein. The vein is now isolated from between the two pleural reflections.

just through the epidermis in the fifth intercostal space, extending it from the posterior axillary line forward practically to the sternum. This knife is now discarded and skin towels are immediately sutured to the skin edges. The incision is then carried through the subcutaneous tissues and muscle layers down to the endothoracic fascia with a second knife. All bleeding points are clamped and ligated. The pleura is then incised keeping close to the upper border of the sixth rib to avoid the intercostal neurovascular bundle. A wide exposure is obtained by the lateral flexion of the vertebral column with the convexity toward the operative side (by means of a folded sheet) along with the use of a

laminectomy retractor. By grasping the individual lobes of the lung with sponge forceps and applying greater traction on the lower lobe, the pulmonary ligament is exposed to view. This is incised close to the pulmonary border up to the lowest pulmonary vein. At this point the vein is isolated from between the two pleural layers, and divided between medium silk sutures. The same procedure is carried out for the middle and superior pulmonary veins. The pulmonary artery is then isolated and ligated proximally with a ligature of doubled braided silk. A tie is then placed distal to this ligature, and the artery is divided between the two sets of ligatures.

Fig. 2.—The pulmonary veins are isolated and divided individually between medium silk ligatures. The pulmonary artery is then isolated and doubly ligated with braided silk. This procedure completely frees the bronchus anteriorly, but does not disturb the peribronchial tissues.

This procedure now frees the bronchus anteriorly but does not disturb the peribronchial tissues. The lobes of the lung are now drawn toward the operator, thus exposing the posterior aspect of the root of the lung. Meticulous care at this point is necessary to preserve the blood supply to the bronchial stump and to prevent injuring the vagus nerve. Each of the two or three bronchial arteries (accompanied by their corresponding bronchial veins) is then isolated individually, doubly ligated with fine silk, and divided at the point at which the bronchus is to be cut across, thereby preserving the blood supply of that part of the bronchus which is to remain. The fibers of the pulmonary plexus of the vagus are then severed. At this point the vagus nerve is gently displaced

posteriorly to free it from the bronchus. This allows for a more adequate inversion of the bronchial stump without interfering with the vagus nerve. Great care, however, is taken not to strip the peribronchial tissues from the bronchus, which is now entirely freed. At this point two rubber shod bulldog clamps are put across the main bronchus above and below the line of incision. The bronchus is then divided with a scalpel, following the line indicated above. Both the scalpel and the resected lung are immediately discarded. If the bronchus is grossly contaminated with foul sputum (as was the case in the animals with experi-

Fig. 3.—The technic for freeing the bronchus posteriorly. Note particularly the ligation of the bronchial vessels along the line of proposed trans-section of the bronchus. This technical detail is necessary to preserve an adequate blood supply to the bronchus and to the peribronchial tissues.

Insert shows the retraction of the vagus nerve (after division of its connecting fibers to the pulmonary plexus) so as to allow for a more adequate inversion of the bronchus without interfering with the vagus nerve.

mentally produced chronic lung abscess), the mucosa of the stump is phenolized followed by alcohol and the first inversion is accomplished by a running Cushing suture of No. 0 chromic catgut. (See Fig. 4 *B*.) If the bronchus is not grossly contaminated, the phenolization is omitted and the first inversion carried out with a running Cushing suture of fine silk. Further inversion is then accomplished by two rows of inverting interrupted mattress sutures of medium silk. All sutures are introduced only through the peribronchial tissues, care being taken never to pene-

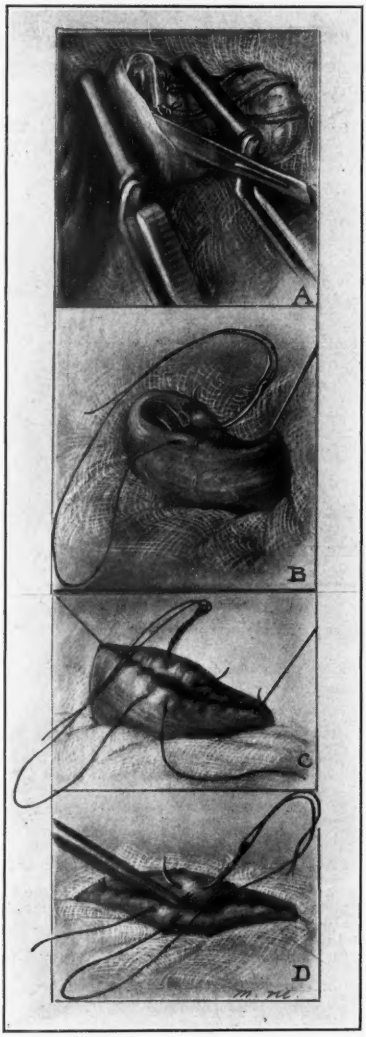

Fig. 4.—The various steps in the treatment of the main bronchus: *A*, the atraumatic division of the bronchus after protecting the pleural surfaces with gauze packs; *B*, the first inversion by means of a running Cushing suture (fine silk or catgut); *C* and *D*, further inversion accomplished by two rows of inverting interrupted mattress sutures (medium silk—Series B, catgut—Series A) applied only to the peribronchial tissues. In *C* the sutures are being introduced from right to left. In *D* the final suture of the last row is being placed. The inversion is more easily accomplished by placing the end sutures first. Gentle pressure applied by means of a hemostat to the ends of the underlying suture of the second row greatly facilitates further closure. Great care must be taken that the sutures do not penetrate the bronchial lumen of a contaminated bronchus.

Fig. 5.—Note the retraction of the inverted bronchus into the mediastinum. The bronchus has just been tested for leakage by flooding the area with saline, prior to closure of the thoracic wound.

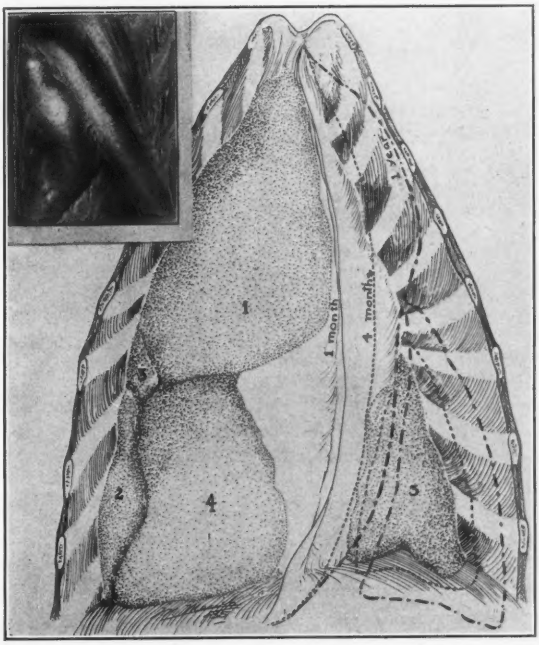

Fig. 6.—Note the gradual shifting of the mediastinal structures toward the operated side, slowly encroaching upon that pleural space, but never obliterating it as a potential cavity. Insert reveals the completely pleuralized stump as a rounded, slightly elevated area four weeks after operation. Note the absence of reaction and of adhesions.

Fig. 7.—Anatomic changes four months after total pneumonectomy on the left. Note absence of gross deformity of thoracic wall in *A*. *B* shows some narrowing of intercostal spaces on the left. *C* shows the compensatory enlargement of the right lung with a shifting of the anterior mediastinum to the left, particularly in the upper chest, while the third lobe pushes the posterior mediastinum ahead of it as it advances to occupy the lower left chest.

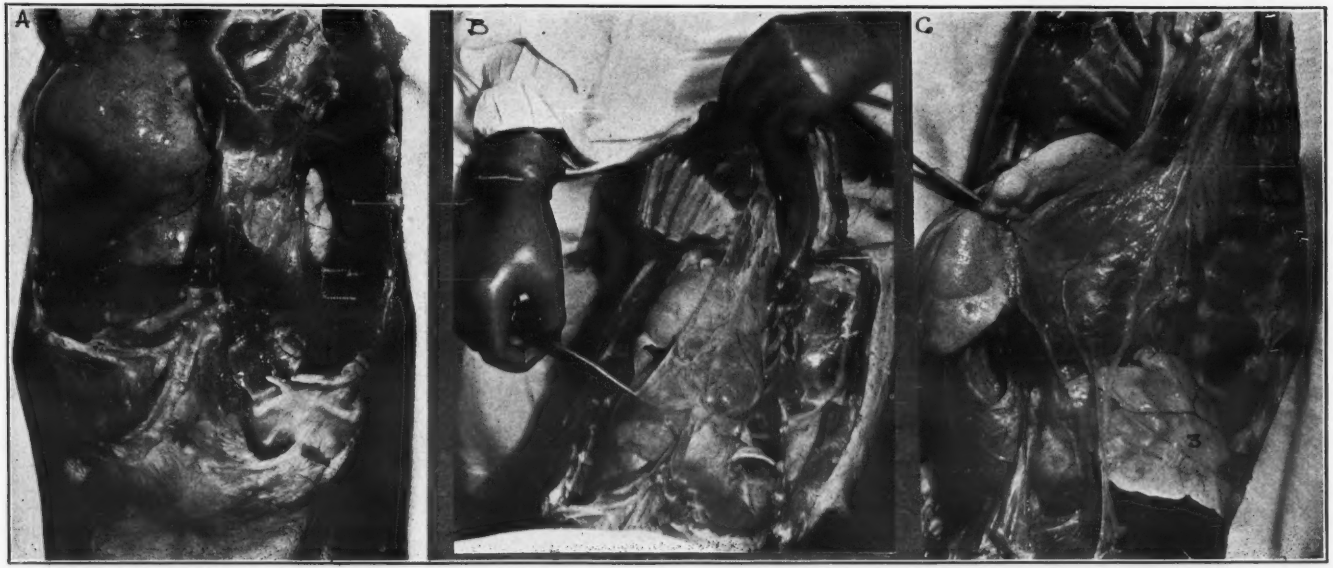

Fig. 8.—Anatomic findings in a dog one year postpneumonectomy. *A* shows absence of any elevation of the diaphragm on left side. *B* shows marked pocketing of both anterior and posterior mediastinum as the left pleural cavity is almost completely encroached upon by the remaining lung. *C* shows clearly the absence of adhesions, the shifting of the lower posterior portion of the mediastinum, and the dimpling of the mediastinum in the region of the bronchial stump.

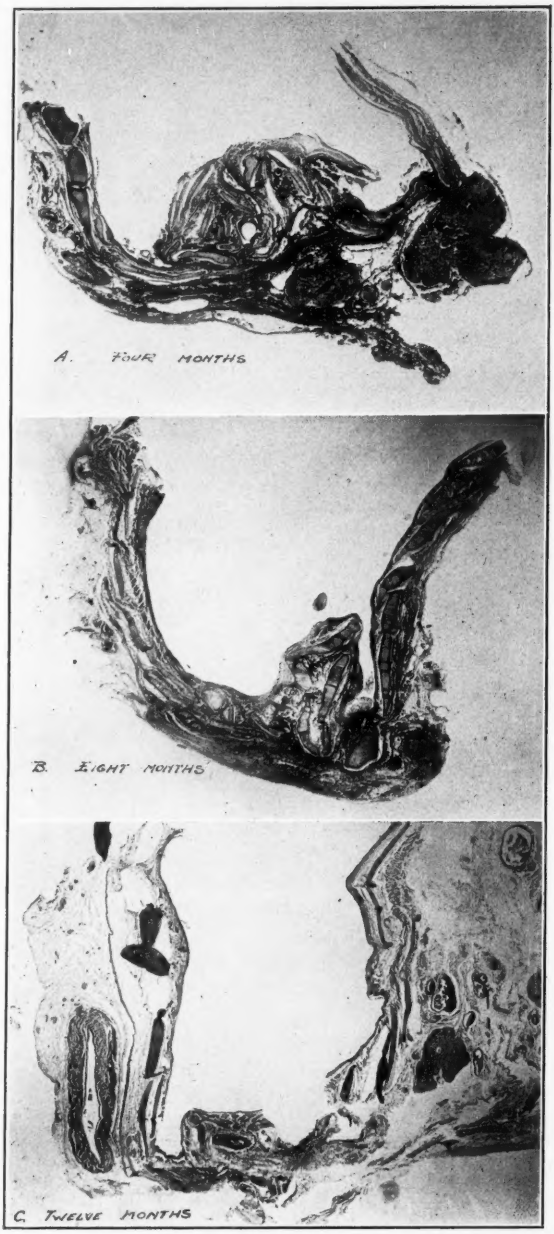

Fig. 9.—Histologic cross-sections of the bronchial stump four months, eight months, and twelve months postpneumonectomy. Disturbances of continuity are due to artefacts in the preparation of the sections.

trate the bronchial lumen. The details of the technic are shown in the accompanying illustrations. The sutures are tied just tightly enough to approximate and not to strangulate the peribronchial tissues. By means of this technic the largest bronchial stump can be gradually and completely closed. After the closure is completed, the bronchial stump is tested for leaks by flooding the line of closure with saline. If the entire procedure is carried out carefully, scarcely 2 c.c. of blood should be spilled into the pleural cavity. After a thorough inspection of the entire pleural cavity, the ribs are approximated by pericostal sutures of braided silk. The muscles are closed in layers with silk, and the skin is approximated with subcuticular sutures.

RESULTS

Prior to the introduction of the above technic 6 pneumonectomies were performed, with ligation en masse of the entire hilum. Of the 6, one animal survived and was sacrificed at the end of one year, 3 died of tension pneumothorax, one of hemorrhage, and one of secondary shock. (For details see Table I.) The mortality rate for this series was 83.4 per cent, while the incidence of bronchial fistulas in animals surviving more than forty-eight hours was 75 per cent.

TABLE I

LIGATION EN MASSE

EXPERIMENT	LENGTH OF LIFE	CAUSE OF DEATH	FINDINGS AT AUTOPSY
		Series A. With Thirty-Day Chromic Catgut (No. 2)	
1. Dog 209	1 day	Acute gastric dilatation and hemorrhage from bronchial artery.	Massive left hemopneumothorax.
2. Dog 311	1 day	Circulatory collapse. Ligatures OK.	Right cardiac dilatation.
3. Dog 313	4 days	Bronchial fistula and tension pneumothorax.	Bronchial fistula. Hilar tissue distal to ligature necrotic.
4. Dog 194	6 days	Bronchial fistula and tension pneumothorax.	Bronchial fistula. Hilar tissue distal to ligature necrotic.
5. Dog 186	1 year	Sacrificed.	Entire left pleural cavity still patent. Stump retracted. Few adhesions.
		Series B. Black Silk	
1. Dog 186	4 days	Bronchial fistula and tension pneumothorax.	Bronchial fistula. Hilar tissue distal to ligature necrotic.
Mortality 83.4%			
Incidence of bronchial fistula in animals surviving more than 48 hours 75.0%			

Greatly discouraged by the results with ligature en masse, the technic described above was carried out in 30 dogs (in some of which the lung was removed because of a chronic experimentally produced lung abscess). In the earlier part of this series employing the new method it was found that those bronchial stumps healed better and more animals

survived in which black silk had been used as the suture material of the peribronchial tissues, regardless of whether the animal had a lung abscess. On the other hand, most of the animals in which chromic catgut had been used as a suture material to the peribronchial tissues developed a bronchial fistula and subsequently died of a tension pneumothorax. Because of these findings it was decided to run two series, in order to determine the possible relation of the suture material to the healing of the bronchial stump. In Series A (12 dogs) in which twenty-day No. 0 chromic catgut was used as the suture material to the bronchial stump,

TABLE II

CLOSURE OF BRONCHUS BY INTERRUPTED INVERTING SUTURES

EXPERIMENT	LENGTH OF LIFE	CAUSE OF DEATH	FINDINGS AT AUTOPSY
Series A. With Twenty-Day No. 0 Chromic Catgut			
1. D. 183	2 days	Hemorrhage from pulmonary artery.	Massive left hemopneumothorax.
2. D. 218	3 days	Bronchial fistula and tension pneumothorax.	Bronchial fistula with marked shifting of mediastinum to right and marked compression of right lung. Small amount serous exudate.
3. D. 308	3 days	Ditto.	Ditto.
4. D. 365	4 days	Ditto.	Ditto.
5. D. 337	6 days	Ditto.	Ditto.
6. D. 367	6 days	Ditto.	Ditto.
7. D. 351	8 days	Ditto.	Ditto.
8. D. 301	10 days	Ditto.	Ditto.
9. D. 255	16 days	Empyema of left pleural cavity	250 c.c. of fibrinopurulent exudate.
10. D. 385	3 weeks	Survived bronchial fistula which developed on fifth day. Sacrificed in three weeks.	Bronchial fistula still present, 50 c.c. purulent exudate. Moderate inflammatory reaction of pleural cavity.
11. D. 369	30 days	Sacrificed.	Pleural surfaces intact. No exudate. No adhesions.
12. D. 314	60 days	Sacrificed.	Pleural surfaces intact. No exudate. No adhesions.
Mortality 75.0%			
Incidence of bronchial fistula 66.6%			

the mortality rate was 75 per cent and the incidence of bronchial fistulas was 66.6 per cent. In Series B (18 dogs) in which fine and medium silk were used as the suture material of the bronchial stump, the mortality rate was 16.6 per cent and the incidence of bronchial fistulas was 5.5 per cent. Except for the difference in material, all details of technic were the same in both Series A and B. Only 3 animals developed empyema, 2 in the catgut series as against one in the silk series in spite of the fact that there was gross contamination of the bronchial stump in many of the animals operated upon.

The surviving animals were sacrificed at various intervals from one day to twelve months to study the healing of the bronchial stump. The

following findings were noted. Twenty-four hours following pneumonectomy the stump was found to be intact but showed slight swelling and moderate injection on gross examination. Microscopically there was noted a dilatation of all peribronchial vessels along with some edema of the peribronchial tissues. There was also noted a polymorphonuclear invasion of the tissues close to the line of closure along with a deposit of fibrin on the surfaces. By the end of seventy-two to ninety-six hours the polymorphonuclear leucocytes had increased in number, and beginning

TABLE III

CLOSURE OF BRONCHUS BY INTERRUPTED INVERTING SUTURES

EXPERIMENT	LENGTH OF LIFE	CAUSE OF DEATH	FINDINGS AT AUTOPSY
		Series B. Black Silk	
1. D. 317	1 day	Acute cardiac dilatation.	Right-sided cardiac dilatation.
2. D. 200	1 day	Sacrificed.	Ligatures intact. No exudate.
*3. D. 374	4 days	Bronchial fistula and tension pneumothorax.	Sutures which had been applied only to the looser peribronchial tissues had cut through.
4. D. 279	6 days	Empyema.	200 c.c. thick purulent exudate.
5. D. 337	1 week	Sacrificed.	Ligatures OK. No exudate. No adhesions. Pleural cavity intact.
6. D. 346	2 weeks	Sacrificed.	Ligatures OK. No exudate. No adhesions. Pleural cavity intact.
7. D. 360	2½ weeks	Sacrificed.	Ditto
8. D. 353	3 weeks	Sacrificed.	Ditto
9. D. 334	3½ weeks	Sacrificed.	Ditto
10. D. 324	4 weeks	Sacrificed.	Ditto
11. D. 366	6 weeks	Sacrificed.	Ditto
12. D. 319	2½ months	Sacrificed.	Ditto
13. D. 325	4 months	Sacrificed.	Ditto
14. D. 310	4 months	Sacrificed.	Ditto
15. D. 193	6 months	Sacrificed.	A few adhesions in costophrenic sinus.
16. D. 208	8 months	Sacrificed.	No adhesions. No exudate.
17. D. 361	10 months	Sacrificed.	No adhesions. No exudate.
18. D. 362	12 months	Sacrificed.	No adhesions. No exudate.
Mortality 16.6%			
Incidence of bronchial fistula 5.5%			

*Sutures intentionally applied to looser peribronchial tissues.

fibroblastic proliferation was noted. The amount of inflammatory reaction observed was far more marked in those bronchial stumps closed with catgut (Series A) than in those in which silk alone (Series B) was used. This reaction was particularly marked in those stumps which had broken down and a bronchial fistula had formed, although the peribronchial tissues themselves were still viable. Grossly, the fistulas varied in size from 2 to 3 mm. in diameter to the size of a wide open bronchus. In many of them the catgut sutures were found lying loose along the fistulous tract. By the twelfth to fourteenth day the line of closure was

noted to be sealed over with a loose network of newly formed granulation tissue. By the end of three to four weeks this had become more dense and the stump consequently more solid. By this time the external surface of the stump was pleuralized and the mucous membrane was starting to cover the inner surface of the bronchial stump. On subsequent studies (two, four, six, or eight months postpneumonectomy) the silk sutures were noted to be surrounded by a fibrous tissue capsule, which contained relatively few round cells and but few foreign-body giant cells. Vestiges of the silk were still present ten to twelve months after pneumonectomy. Not only the peribronchial tissues in the stump inverted by this method but the cartilages as well were found to remain viable in practically all instances. In only a few stumps was there noted a slow breaking down of the inverted cartilage.

In the animal which survived ligation en masse the cartilages beyond the point of ligation had completely degenerated and were replaced by fibrous tissue. The mucous membrane completely covered the inner surface of the stump by the sixth to eighth week.

As to the stump itself, it was found to retract into the mediastinal tissues so that the stump, which could be identified as a slightly rounded elevation three to four weeks postpneumonectomy, could by the fourth to sixth month be noted only as a dimpling of the mediastinal pleura and its outline alone determined by palpation.

As to the fate of the pleural cavity—in none of the animals sacrificed following pneumonectomy in which closure of the bronchus was carried out with silk, was there any exudate found. Adhesions, also, were conspicuous by their absence. The pleural cavity on the pneumonectomized side remained a potential cavity, in spite of the fact that it was encroached upon by the gradual shifting of the mediastinal structures. By the end of one year, as demonstrated by roentgenograms and as seen at autopsy on animals following fixation of the organs in situ with formalin, the space was so encroached upon by the enlarging lung of the opposite side as to be occupied completely except for a small area at the apex and in the costophrenic sulcus. In spite of this the pleural space was still intact, and but few adhesions between the mediastinal and costal pleural surfaces were noted. These findings closely simulated those described by Heuer and Dunn.[22]

SUMMARY

Review of the literature not only of experimental lobectomy and pneumonectomy, but also of the clinical fields as well, reveals that obtaining a completely air-tight bronchial stump is the most difficult as well as the most important phase of the problem. The high incidence of bronchial fistula along with the high incidence of pleural infection are to be ascribed essentially to the inherent difficulties in the care of the bronchial

stump. The inherent difficulties are to be explained on the basis of the singular anatomic structure of the bronchus with its cartilaginous rings and meager blood supply. The healing of the bronchial stump is brought about by the peribronchial tissues. Any interference of the meager blood supply to these tissues by rough and careless handling of the stump will either impede healing of the bronchial stump or lead to necrosis with the inevitable development of a bronchial fistula. The fact also that the healing is brought about only by the peribronchial tissues, the blood supply to which is meager, indicates very clearly that the reparative process must needs be slow and that a considerable time must elapse before the closure of the bronchial stump may be regarded as solid. The fact that the mucous membrane of the bronchial tree is frequently contaminated with buccal organisms, requires that no suture used penetrate into the bronchial lumen. Therefore, the ideal technic for the closure of the bronchial stump will be that method in which meticulous care is taken not only to prevent infection but also to minimize the trauma to the bronchus along with avoiding any disruption of the normal continuity of structures about the hilum.

The technic as described by Willy Meyer meets most of these requirements but has, we believe, several distinct disadvantages. In the attempt to overcome these disadvantages enumerated above, certain modifications were made and this technic was carried out in thirty animals (dogs). The technic requires the separate treatment of blood vessels along with an atraumatic division of the bronchus followed by the closure of the bronchial stump by means of inverting Cushing and mattress sutures. Considerable emphasis has been placed upon the preservation of the blood supply to the peribronchial tissues (for details see accompanying diagrams). The sutures are introduced only through the peribronchial tissues, care being taken never to penetrate the bronchial lumen. The sutures are tied only tight enough to approximate and not to strangulate the tissues. By means of this technic the largest bronchial stump can be gradually, easily, and completely closed.

Prior to the introduction of the above technic six pneumonectomies were performed with ligation en masse of the entire hilum. The mortality rate in the series was 83.4 per cent, while the incidence of bronchial fistulas in animals surviving more than forty-eight hours was 75 per cent. Greatly discouraged by the results with ligature en masse the new technic was developed and was carried out in 30 dogs (in many of which the lung was removed because of a chronic experimentally produced lung abscess). Because of the high incidence of bronchial fistulas in the earlier work in which the stumps were closed with catgut, which stood out in sharp contrast to the almost perfect healing following silk closure, it was decided to run two series (A and B) for which all details of technic would be the same except for the suture

material. In Series A (12 dogs) in which twenty-day No. 0 chromic catgut was used as the suture material of the bronchial stump, the mortality rate was 75 per cent and the incidence of bronchial fistula was 66.6 per cent. In Series B (18 dogs) in which fine and medium silk was used as the suture material of the bronchial stump, the mortality was 16.6 per cent and the incidence of bronchial fistula 5.5 per cent.

Histologic examination of the bronchial stumps at various stages of the healing process reveals marked inflammatory reaction to catgut buried in the peribronchial tissues, in sharp contrast to the slight reaction to the silk suture material. Silk is subsequently tolerated remarkable well in the peribronchial tissues, and vestiges are still present at the end of one year. It soon becomes encysted in a fibrous tissue capsule and so it remains, provided care has been taken to prevent its penetration into the bronchial lumen.

The healing was in all cases brought about by the peribronchial tissues, although this was sluggish in contrast to the rate of healing seen in other tissues. It is felt that the advantage of silk as so evident in the experimental work lies in the fact that it is less irritating to tissues and that its tensile strength is not easily affected, and, consequently, it holds the peribronchial tissues in apposition during the relatively long period required for the solid repair of the bronchial stump.

CONCLUSIONS

1. The method of Willy Meyer, which was modified in the attempt to apply it to the treatment of large bronchi, allowed for the easy closure of the main bronchus in all cases.

2. Silk suture material is tolerated very well in the peribronchial tissues as long as care is taken not to allow the suture material to penetrate the bronchial lumen when the latter is grossly contaminated.

3. The low incidence of complications following the use of silk as the material in the closure of the main bronchial stump, in contrast to the high incidence of bronchial fistulas following the use of catgut in a control series, suggests the use of silk for the closure of the bronchial stump in human beings.

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THE OPEN INTRAPLEURAL PNEUMOLYSIS OPERATION*

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THE intrapleural pneumolysis operation has become a valuable adjunct to our collapse therapy measures. The closed method described by Jacobaeus[1] in 1915 has received the most favor, and Moore[2] in a recent review of the literature was able to collect 2,000 cases of closed operation and only 55 instances of open operation. O'Brien,[3] Eloesser,[4] Bettman,[5] Sauerbruch[6] and others have opened the chest wall to sever limiting adhesions, but technical difficulties or the development of complications have discouraged the general adoption of this method.

In our early experience, with inadequate apparatus for the closed procedure, we first found that the pleural cavity could be opened, when a pneumothorax was already established, with little or no respiratory disturbance. We then recognized that we could cut adhesions with more confidence than formerly and began to attack larger bands, hitherto deemed inaccessible.

At present we feel we have standardized our technic and management so as to make the open operation a safe procedure, applicable for general use in severing limiting adhesions.

The adhesions which require cutting are those which definitely hold cavities open in spite of continued treatment (Fig. 1). Often bands will not prevent collapse of the cavities (Fig. 2), and only a small percentage (in our clinic approximately 5 per cent) require surgical consideration.

Preoperatively, the adhesions are carefully studied with the aid of fluoroscopy and stereoscopy to determine their extent and position. The actual picture is almost always much more complicated than the x-ray plate would lead one to believe, for in an end view a large band may appear as a narrow string and thin webs may not cast a visible shadow.

The equipment necessary is only the usual surgical instruments supplemented by a small stick light and a long stem cautery. Electrocoagulation, if available, is desirable, but the ordinary electrocautery will suffice.

Local infiltration of 1 per cent novocaine has afforded adequate anesthesia, and apparatus to maintain differential pressures has not been necessary in any of our operations.

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The explanation of the fact that the pleural cavity can be opened widely when the lung has been collapsed by pneumothorax without occasioning any marked respiratory embarrassment has not been satisfactorily given. Thickening of the pleura and fixation of the mediastinum are probably a factor, but it is hard to see how the mediastinum could be so affected after a short period of treatment. We

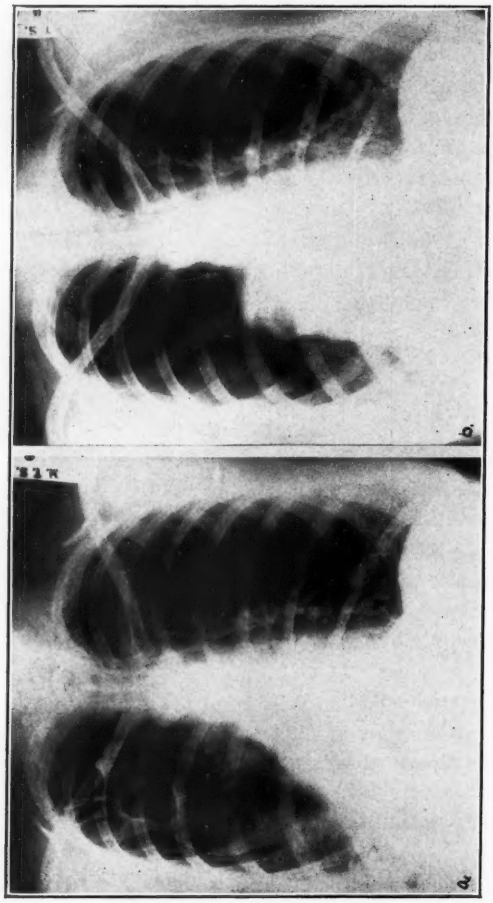

Fig. 1.—Case in which adhesions required cutting. *A*, Before operation (retouched); *B*, after operation.

are extremely cautious in opening the pleural cavity in patients with a very lax mediastinum, but the only case of respiratory disturbance occurred where there was very little previous collapse of the lung.

An incision is made in the axillary line (Fig. 3), this approach being advantageous because few tissues are transgressed; the opening enables us to reach the anterior and posterior surfaces of the lung, and the result-

ing scar is obscure. The interspace to be selected will depend upon the location and extent of the adhesions previously determined as accurately as possible. We most frequently approach through the third interspace, as this entrance enables us to reach the apex, and we find this defect more easily closed than one in the second interspace. The inter-

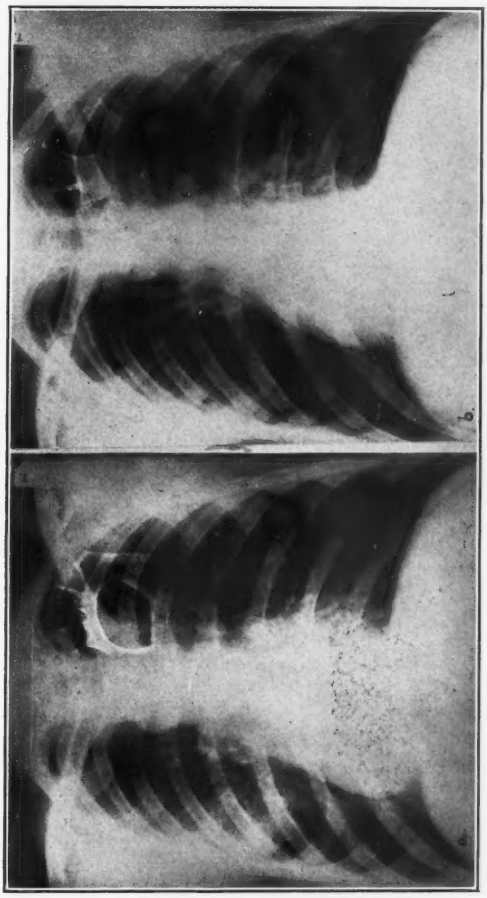

Fig. 2.—Instance in which adhesions have not prevented closure of cavity (retouched). *A*, Cavity open; *B*, cavity closed. Patient well and working.

costal structures and the pleura are incised midway between the ribs; an opening two to three inches long usually affords ample exposure.

With the aid of the stick light, used particularly for transillumination, we can now determine the extent of the limiting bands and the feasibility of cutting them. The bands are usually easily severed with the electrocautery or the endothermic knife (Fig. 4). Close inspection is made for bleeding points, for hemorrhage into a free cavity might easily be disastrous, and any bleeding points can be controlled by crush-

ing, ligation, or coagulation. Blood or fluid in the pleural cavity should be removed, because it may form a suitable culture medium for infection, and it is our practice to lavage thoroughly the pleural cavity with a warm saline solution at the end of the operation.

Fig. 3.—Position on table and incision for open intrapleural pneumolysis operation.

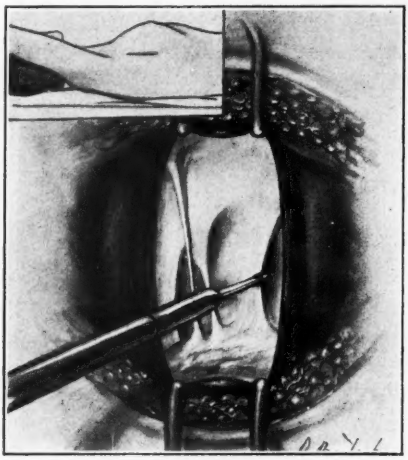

Fig. 4.—Cutting of adhesions with endothermic knife through intercostal opening in chest wall.

The closure is the most difficult part of the operation, and it is of utmost importance that an air-tight union of the pleural margins be obtained. Approximation of the edges by kangaroo tendon or heavy catgut sutures through or around the adjacent ribs supplemented by a few interrupted sutures in the pleura and intercostal tissues gives the

best results (Fig. 6). It has occasionally been necessary to cut and retract one rib to reach a difficult adhesion, and this procedure may also be used to facilitate closure. The soft tissues are then sutured and a heavy dressing is applied, extreme care being taken to avoid contamination at the time of operation and through the succeeding days.

POSTOPERATIVE CARE

Postoperatively, the head of the bed is slightly elevated, and the patient is turned toward the unoperated side to prevent fluid from interfering with the primary union. It is necessary to give the first air refill within a day or two, and the amount of collapse should be watched very care-

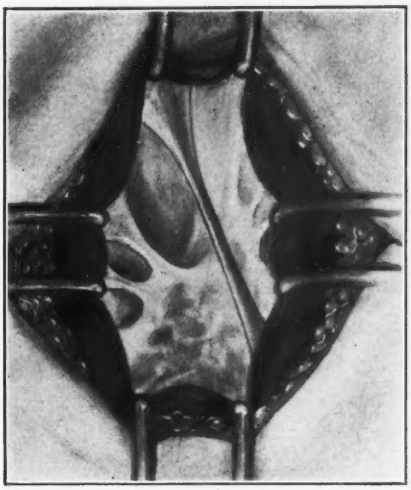

Fig. 5.—View of complex adhesions through window in chest wall.

fully during the period following. Some subcutaneous emphysema usually will be present, but this invariably gradually subsides if the closure has been adequate. Some effusion will result in a large percentage of cases, and the success of the operation largely depends upon the proper management of this condition. We remove this fluid through a needle on the second or third day and wash out the pleural cavity with a warm saline solution. This procedure is repeated until there is no further tendency for fluid to reform; usually one to two aspirations suffice. If the fluid is allowed to remain, it not only occasions a severe constitutional reaction but also forms an excellent medium for infection and frequently favors the development of empyema.

The discovery at the time of operation of a tuberculous pleuritis forbodes the development of an empyema, but this, of course, cannot be attributed to the operation.

Subsequently, there will be other problems such as are associated with pneumothorax treatment in general. As a result of these effusions the pleura is thickened and reexpansion of the lung may be difficult to obtain. Then oleothorax or thoracoplasty may come into question.

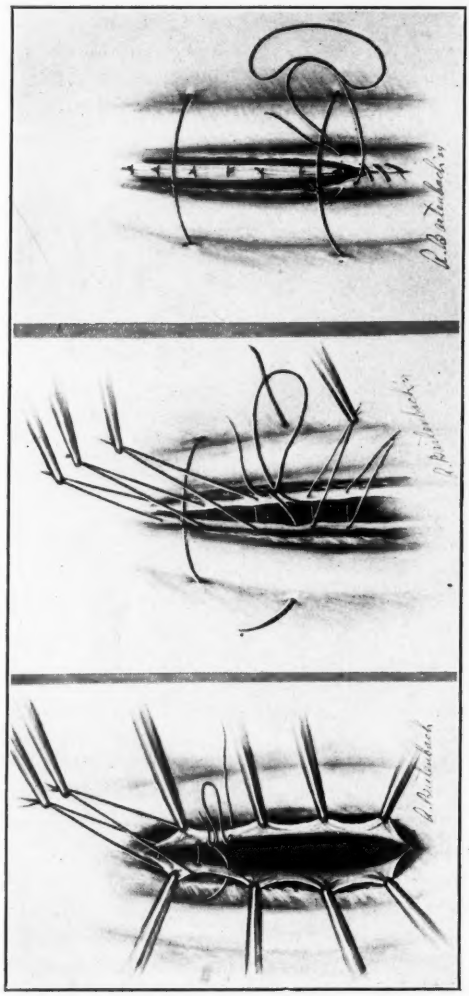

Fig. 6.—Steps in closure of intercostal opening. *A*, Interrupted sutures being placed in the parietal pleura; *B*, kangaroo tendon sutures placed around adjacent ribs; *C*, pleural sutures tied after approximation of ribs, continuous suture being made through intercostal muscles and fascia.

RESULTS

We have performed the open operation 44 times. At first we encountered some difficulties in approach and closure which had to be overcome. Anterior, posterior, and lateral approaches were tried, and in

most cases a section of rib was removed leading to difficulty in closing the defect. Severe postoperative reactions occasionally occurred from the resulting effusions, and until we controlled these by aspiration of fluid and irrigation of the pleural cavity it seemed that we would have to abandon the operation. Consequently, we feel that it is only fair to divide the cases into two groups: (1) those performed before January 1, 1934, and (2) those performed since that date in which the operative technic and postoperative care have been exactly the same. (Table I.)

TABLE I

RESULTS OF OPEN PNEUMOLYSIS OPERATIONS

	GROUP I PATIENTS OPERATED BEFORE JANUARY 1, 1934	GROUP II PATIENTS OPERATED JAN 1, 1934–NOV. 1, 1934
Technically and clinically successful	10	15
Technically successful, clinically unsuccessful	2	3
Technically unsuccessful	8	2
Complications of fistula and empyema	3 All subsequently died	1 Progressing favorably 4 months after
Total	23	21

In those cases classified as technically and clinically successful, the adhesions were cut, the cavity was closed, and there were no complications other than temporary effusions.

In those cases recorded as technically successful but clinically unsuccessful, the adhesions were cut and there were no complications, but for various reasons the cavities were not closed. In those grouped as technically unsuccessful the adhesions could not be cut but no complications resulted.

The serious complications all developed in the same manner; inadequate closure or infection led to pleurocutaneous fistula and empyema. One of these patients died of general toxemia, the second of myocardial failure one month after completion of a thoracoplasty; a third died of tuberculous meningitis. In the most recent case the patient is progressing fairly well at present and having very little absorption from the empyema cavity which, of course, must eventually be collapsed.

SUMMARY

Intrapleural pneumolysis is not a procedure to be undertaken lightly. It should be performed only when necessary and then with extreme caution. The closed method is satisfactory for severing the more simple bands, but the open method is, we feel, safer and more efficient where multiple and more complex adhesions are present.

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THE MORTALITY OF EMPYEMA*

An Analysis of One Hundred Consecutive Deaths From the Records of Charity Hospital in New Orleans

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ALTHOUGH many writers on the subject have made the statement that the lessons of empyema have been purchased at a great price, few of them are rash enough to assert that those lessons have been learned. Indeed, the most most casual survey of the literature proves quite definitely that they have not been. The principles of the treatment of empyema are perfectly well established, but rare is the surgeon who does not on occasion, sometimes on many occasions, persuade himself that the case presently under his care is one which is an exception to the rules. Part of the confusion, too, undoubtedly arises from the fact that particularly since the War there has been a general tendency for the principles of the treatment of empyema to be lost sight of under a mass of details; it would be easy to assume, after reading some contributions to the subject, that the evacuation of the pleural cavity was by no means as important as the gadgets which the authors of the papers had devised to accomplish that end. Here, as elsewhere, the trees too often conceal the wood.

The delusion that one's results are better than they really are is a very common failing and a failing that is not confined to medicine. The truth is often a shock to one's complacency, but shocks of such a sort are always salutary, and for that reason it is our custom to set ourselves at intervals to an analysis of our hospital records, in the belief that this is a particularly accurate method of arriving at the true facts of whatever disease we happen to be investigating. Furthermore, the results achieved by many surgeons in a large series of cases are usually a far better cross index of the situation than are the results of an individual surgeon; they may be less brilliant, but they are likely to be more enlightening.

The general facts of empyema in Charity Hospital in New Orleans were excellently expounded a few years ago by Ochsner and Gage, in a review of one hundred consecutive cases, and a further contribution of that sort would be superfluous at this time. But a survey of the

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medical literature of the past several years has revealed to us that there has been no recent publication specifically devoted to the mortality of empyema, with the exception of Muller's paper in 1931, nor, so far as we are able to discover, has there been for many years any publication dealing solely with an analysis of fatalities. For that reason we have chosen to investigate one hundred consecutive deaths from empyema from the records of Charity Hospital, and we have analyzed them not so much statistically as with the idea of determining, so far as it is possible after the event, why they occurred and what could have been done to prevent them.

In order to comprehend the part, however, it is necessary to survey the whole, and we therefore, as a background, investigated the empyema admissions in Charity Hospital for the last ten years, as well as the admissions for influenza and for pneumonia, the two conditions which most often precede it. For one thing, we were very keenly interested in the point made by Heuer, that the seasonal variation in virulence has more to do with the mortality of empyema than has the particular method of treatment adopted, provided, of course, that the method conforms to certain general principles. The incidence of respiratory affections in this part of the country is relatively low, for our climate is always temperate and frequently tropical; there is an abundance of fresh air and sunshine, and there is a notable absence of crowding from overpopulation. As a result, no physician has a very extensive experience in empyema; in the 100 deaths we studied are represented a total of 58 physicians, including 4 pediatricians, 12 internists and 42 surgeons. As a further result, the disease is treated by a bewildering variety of methods and variations of methods. It seemed to us that here, if anywhere, would be the place to prove or disprove Heuer's thesis that treatment is a comparatively secondary consideration to the virulence of the infection in certain years and at certain seasons.

That we were not able to prove it Fig. 1 shows very clearly. In the ten-year period studied there were 2,979 cases of influenza with 137 deaths, a mortality of 4.6 per cent. There were 6,056 cases of pneumonia with 2,370 deaths, a mortality of 39.1 per cent. And there were 682 cases of empyema with 117 deaths, a mortality of 17.1 per cent. The incidence of empyema after pneumonia, therefore (its incidence after influenza is slight unless influenza happens to be really epidemic, as it was in 1918), is 11.2 per cent, which is approximately what it is elsewhere, though the incidence of pneumonia in proportion to the hospital population, which totaled over 235,000 patients during this period, is decidedly low.

But there is not, as in Heuer's study, any correspondence between the yearly mortality of the three diseases. Rather there are apparent a decided number of discrepancies (Fig. 1), the most striking of which is

that in 1926, when the pneumonia mortality was within a fraction of its highest level, the empyema mortality was, with a single exception, at its lowest level. In other years the mortality of one disease rose as that of the other declined, or the increase or decrease in the mortality, even though similar in tendency, was not proportionate; but in no other year was the discrepancy so marked. We went to the original records, therefore, in an endeavor to prove what, we admit, was at first little more

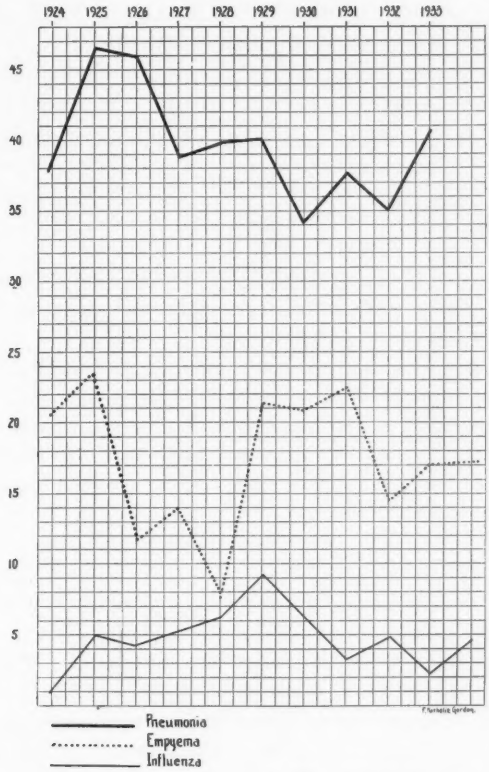

Fig. 1.—Yearly mortality for ten-year period.

than a guess: that in that particular year the pneumonia was apparently of so severe a type that people died of it before they developed empyema. The records seem to substantiate the assumption, for we found that the patients who died of pneumonia in that year were ill on an average only 8.5 days, which includes the duration of illness before admission as well as in the hospital. It is perfectly possible, of course, for empyema to develop within so short a period, but as a rule, unless it is of the synpneumonic variety, it does not. Our records for other years bear out

that statement, and our explanation of the discrepancy in the mortality rates of the two diseases for 1926 is perhaps not as wild as it first seemed.

We noted, next, the very marked discrepancy between the incidence and the mortality of influenza, pneumonia, and empyema in the colored and white races. In the 124 cases of empyema studied from this standpoint by Ochsner and Gage in 1930, the mortality among negroes was 25 per cent against 10 per cent, the mortality among the whites; in our study the disparities are even more marked. We found that in the years from 1924 through 1928 the negroes exhibited a much higher incidence of pneumonia, both absolutely and proportionately, than the whites, but a much lower death rate. In 1926, the year whose contradictions we have already discussed, the mortality was 10 points lower than in the white race. But after 1929 the negro incidence fell nearer and nearer to the white incidence, and in 1931 and 1933 it was actually lower, while the death rate constantly exceeded the death rate among the white patients, in 1933 by almost 10 points. Since the actual proportion of negro to white admissions has remained very much the same over the ten-year period, we have no explanation whatsoever to offer for this reversal of statistics.

We likewise have no explanation to offer of the racial discrepancies apparent in the influenza statistics. Even in the years when the disease was mildly epidemic (everything, of course, is relative compared with 1918), the negro incidence was from one and a half to three times lower than the white incidence, but the death rate was always higher. In one year it was three times as high.

Finally, the relative white and negro incidence of empyema demands some analysis. The negro incidence is very much smaller than the white. In proportion to the hospital pneumonia admissions it is 5.6 per cent against 18.3 per cent, although the negro mortality over the ten-year period is almost three times as high as the white, 30.5 per cent against 11.3 per cent. Evidently the negro, although he seems to develop empyema less frequently than his white brother, is less capable of withstanding its ravages, a point which Ochsner and Gage also emphasize in their consideration of the disease in the two races.

First, from the standpoint of race, we found that 47 of the 100 deaths we selected for analysis had occurred in negroes, which is only slightly higher than the percentage of negro admissions to the hospital. But when these deaths were studied from the standpoint of age, a marked racial difference immediately became apparent. Of the 34 deaths in children under twelve years of age, only 7 were in negroes; but of the 64 deaths in adults, 40 were in negroes, as were 35 of the 55 deaths among adult males. Studies such as this are valuable, as we have said, in that they focus one's attention upon one's own problems, and we

realized at this point that the situation made plain in these figures precisely paralleled our own experience in this disease. A check of our records showed that in the last eighteen months we had not done a single thoracotomy upon a negro child, but that we had done a fair number in negro adults, with rather discouraging immediate and end-results. The negro child, for some reason, seems singularly resistant to empyema while the negro male, in proportion to the total number of cases, seems singularly prone to develop the disease and to develop it in a fatal form.

As a preliminary to the discussion of the causes of death in these cases (Table I), it should be said that sixteen of the patients, 16 per cent of the total number, died in from six to seventy-two hours after admission to the hospital. In four cases, it is true, emergency and probably ill-advised thoracotomies were performed immediately upon admission; but the remaining fatalities in the group can scarcely be charged against either the surgeon or his plan of treatment, for a patient with empyema who dies so promptly after hospitalization is obviously moribund when he is first seen.

That forty deaths in the series, ten of them proved by postmortem examination, were due to toxemia and exhaustion seems at first glance somewhat surprising in view of the rather general belief that empyema per se has a very low mortality and that it is doubtful, in properly treated cases, whether there can be enough absorption to cause fatal toxemia. That all of these patients were properly treated is, of course,

TABLE I

CAUSES OF DEATH IN 100 CONSECUTIVE CASES*

Toxemia and exhaustion	40
Pneumonia	16
Shock	11
Cardiac dilatation and embolism	6
Tuberculosis	5
Kidney disease and complications	4
Pericarditis	4
Pulmonary edema or respiratory failure	3
Rupture of bronchus	3
Gangrene of lungs	2
Meningitis	2
Peritonitis	2
Hemorrhage (ruptured aneurysm)	1
Multiple abscesses of lungs	1

*Thirty-nine proved by postmortem findings.

a preposterous assumption. Some of them, as we have just said, were seen too late for treatment to be of any avail. In one case the diagnosis, for reasons which are not apparent from the record, was not made until after death. In certain other cases the treatment was not such as we ourselves should have chosen to continue indefinitely. But in twenty-one cases thoracotomy was done according to all the accepted rules, and still drainage was inadequate. The distribution of the deaths from this

cause is rather interesting. In this group appear nineteen of the thirty-four children in the series, 55.9 per cent of the total number, but only 32.6 per cent of the adults, which seems to prove that children are far less fitted to endure a long-continued septic process than are older persons. Almost 50 per cent of the white patients fall into this group, too, against less than 30 per cent of the colored, which seems to suggest the same sort of conclusion.

Pneumonia, the next most frequent cause of death, was responsible for nine deaths in adults and for seven in children, two of the latter dying within eighteen hours of admission and therefore not to be charged fairly against the hospital mortality. The patients who were treated by preliminary aspiration, or by preliminary aspiration and later thoracotomy, were treated, so far as we can determine, according to accepted principles; but in five patients in this group emergency thoracotomies were done while the pneumonic process was still active, and, although that procedure can sometimes be done with impunity, it frequently cannot, as these fatalities prove. In one case pneumonia recurred on the same side as the original disease; in seven others it recurred on the opposite side and was the immediate cause of death.

The eleven deaths from shock prove again a point on which we have always been particularly insistent—that the rapid removal of large amounts of pus from the pleural cavity, no matter whether it is done by aspiration or by surgical measures, is in direct violation of all the laws of physics and physiology. The chest is not a region in which liberties can be taken. The human organism does not tolerate insults to its respiratory apparatus any more than to its circulatory apparatus, and the rapid evacuation of fluid collections from the pleural cavity was unquestionably responsible for at least some of this particular group of deaths. It is our own policy in such cases to use a technic that we grant is crude but that does provide for what we may term "delayed open drainage." On the operating table the chest is opened, and drainage tubes are inserted, but nothing more is done. We make certain that we have made the opening into the pus collection, but under no circumstances do we suction the fluid out. The tubes are then covered with large gauze pads, which are not disturbed for at least eight hours. As the gauze becomes soaked with pus, it controls in a crude sort of way the rate of evacuation, and our patients are rarely shocked after thoracotomy if their condition is such that they can withstand the operative act at all.

Certain of the other groups of cases demand a word of comment. In the six cases in which death was attributed to cardiac dilatation and embolism, four of them proved by postmortem examination, all of the patients had some underlying cardiac complication which made the outcome doubtful from the beginning; all of them were adults, and five were colored males, as were the five patients who died of tuberculosis.

Two of the four deaths from pericarditis were proved by postmortem findings; and, although in the other two cases the diagnosis is only presumptive, the pictures are classical. In three of the four patients who died of kidney complications, the kidney pathology overshadowed the chest condition. One child, for instance, was admitted with an extreme degree of anasarca, and these cases, together with another case in which death was due to peritonitis following a perinephritic abscess and in which the empyema was not discovered until postmortem, seem to prove again that the coexistence of widely dissimilar diseases is not incompatible. Obviously a patient with any sort of disease can develop empyema, just as a patient with empyema can develop any other disease. One condition is no guarantee against the development of another, and eternal vigilance is the price of safety.

In the three cases listed as rupture of a bronchus, the sequence of events seems quite clear. Two of the patients were being treated by aspiration and air replacement, and the third, a child, had had an open thoracotomy following an unsuccessful attempt at closed drainage. In all three cases there was a sudden and violent attack of coughing, followed by an outpouring of purulent fluid through the mouth and nose, spasmodic choking, and almost immediate death. No autopsy was permitted in any case, but there seems little doubt that the patients actually drowned in their own secretions.

The hemorrhage which followed a ruptured aneurysm was brought about by the empyemic condition. The patient, after two previous thoracotomies, had returned with an *empyema necessitatis* for which a third thoracotomy was done. A particularly violent spell of coughing preceded the hemorrhage which caused death. We might say that the absence of cerebral abscess as a cause of death seems rather unusual in so large a series of fatalities, but it could scarcely have been overlooked if it had been present, for thirty-nine of the one hundred cases were submitted to autopsy; and in the remaining sixty-one cases, the causes of death are clearly apparent from the symptom-complex, physical findings, and clinical course, even where they are not specifically stated.

It is true that in all the patients who had thoracotomies done while their pneumonic process was active, respiratory and cardiac action were both seriously embarrassed. The removal of fluid by aspiration would, in our opinion, have been an equally effective and a much safer procedure. Indeed, certain cases in this series of deaths ought to convince the greatest skeptic that the patient's best interests are never served by an emergency thoracotomy. Even if he survives it, as he very frequently does not, he would have been more safely treated by another method. The patient with active pneumonia gains nothing by such a procedure, nor does the patient who is debilitated by his disease, who is frequently critically ill and in a state of shock, and who stands to lose everything by being thrown into a state of deeper shock by a drastic operative pro-

cedure. The dangers of shock in empyema are so clearly recognized that many surgeons advocate operating on the patient in his bed, and the sudden death of one patient in this series, as he was being lifted to a roller for transportation to the operating room, serves to emphasize that point.

On the question of open versus closed drainage much has been said and probably much more could be said, but the matter does not seem to us one of extreme importance, though we do agree with Ochsner and Gage, that perhaps in an institution in which medical and nursing supervision is necessarily limited, open drainage gives the best results. Concerning aspiration, however, much needs to be said, for it is a misunderstood and a misapplied method. The value of diagnostic aspiration is, of course, beyond dispute. No one can gainsay Osler's instructions

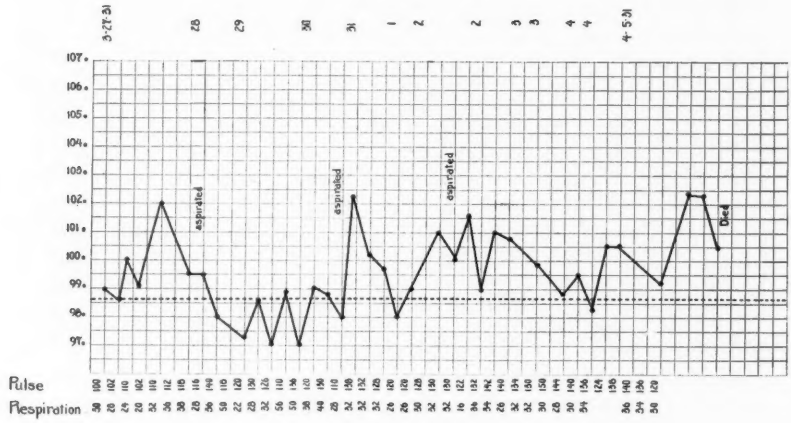

Fig. 2.—Typical chart of a patient treated by aspiration alone.

to his interne, that in presumptive empyema the exploring needle should be inserted in seventy times seven places before the diagnosis is ruled out. Had that rule been observed in some of these cases, we have no doubt that many of the patients would still be alive who died without surgery because the diagnosis, even though suspected, was not established. And in the same connection we might add that we endorse Ashhurst's dictum, that medical pus is quite as good as surgical pus as an indication for operation.

But after diagnostic aspiration, what? There can be no doubt of the value of repeated aspirations in tiding the patient over the critical period of his illness, and in taking care of his cardiac and respiratory embarrassment until the pneumonic process has subsided, the mediastinum is stabilized, and surgery can be done with a minimum risk. But we cannot agree that aspiration as the sole method of treatment for

empyema is a safe or a successful plan. It may be as dangerous as immediate thoracotomy. It frequently permits the optimum time for surgery to slip by, the patient steadily losing ground because of toxemia from the retained and incompletely emptied purulent collection within his chest (Fig. 2). The children in this series who died of toxemia and exhaustion with aspiration as the only method of treatment and the adult who died from the same cause after fifty-one days of treatment by it are evidences of such mischances. If aspiration is done, says Muller, it should be done in terms of days, not of weeks. The wisdom of that injunction many cases in this series prove. Young, in his Lumleian Lectures on the chest, mentions one surgeon of an ancient day who over a period of nine months removed 30 pints of pus by 74 punctures, and another who made 122 punctures of the chest in eleven

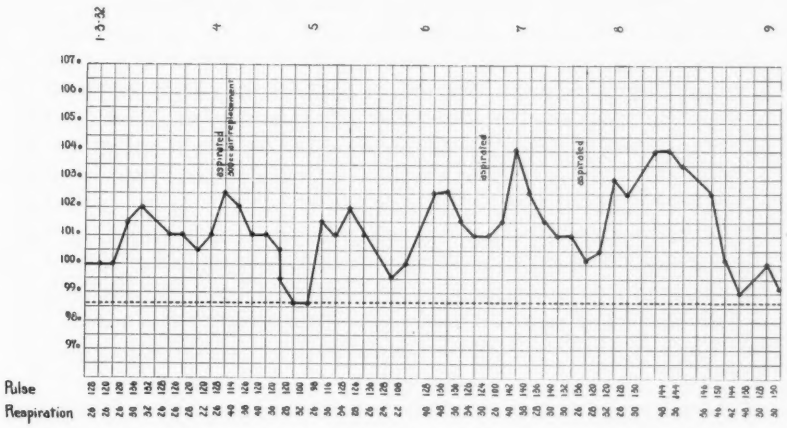

Fig. 3.—Chart of a patient treated by aspiration and air replacement.

months. There were not quite such extreme instances in the group we studied, but some of the cases did bring to our minds those two long-suffering patients. The incomplete evacuation of pus would not be tolerated anywhere else in the body, and it is hard to understand why such a halfway measure should be considered adequate within the pleural cavity.

It must be remembered, too, that reports of cures by aspiration must be scrutinized carefully before they are accepted as such. McEnery and Brennemann's report of a series of children treated by aspiration with a 6 per cent mortality is unusually good, but the authors themselves are the first to point out that it is merely a preliminary report. Moreover, as many writers have emphasized, all cures by aspiration are not cures of empyema: pneumonia is frequently associated with a pleural effusion which, even without treatment, subsides spontaneously

and does not go on to suppuration. In compiling statistics, as Allen says, empyema should be empyema, and thin fluid, even if it is contaminated, does not fall into that category .

Aspiration with air replacement, which was devised independently by Elias and by Danna, has given in their hands excellent results; but in ten cases in this series it was the only method of treatment, and in four others it was used until its inadequacy was apparent and thoracotomy was done as a last resort. The wisdom of the selection of certain of the cases is doubtful; one of the patients, for instance, had a grave heart lesion, and three others had tuberculosis. In one instance the removal of 1,500 c.c. of pus at one sitting from the chest of a nine-year-old child is not so much a reflection on the method of treatment as on the surgeon who paid so little heed to the laws of physics. In another case so much air was injected into the chest that the breathing was more embarrassed, and the heart, according to the x-ray pictures, was more displaced than it had been before treatment; the air had to be removed, and this case must be set down as an error on the part of the surgeon rather than as a defect in the method. In case after case in which this form of therapy was used, as the specimen chart (Fig. 3) shows, the reduction in the temperature was negligible, and the clinical progress made it clear that the suppurative process had been incompletely evacuated. Five deaths from toxemia and exhaustion must be charged against the method, which seems, in spite of the excellent personal results of its authors, rather unsafe in the hands of less careful surgeons. Perhaps the solution, as Beck says in another connection, is that if one desires to use another man's method, one should follow it to the letter and not introduce modifications of one's own.

Probably no series of cases of empyema has ever been reported in which the diagnosis has not been occasionally overlooked. Frequently the error arises because the condition is considered to be unresolved pneumonia, a diagnosis which should never be made until Osler's "seventy times seven" rule has been strictly observed. Frequently it arises because, as we have already pointed out, the chest pathology is overshadowed by urgent symptoms of another disease. All too often the empyema is not discovered because the physician or the surgeon fails to look for it. Undoubtedly the surgeon was directly responsible for many of these deaths, but, with no attempt to excuse ourselves for our own shortcomings, it is only fair to say that in others he saw the patient too late to hope that the measures he instituted would be of any avail. In empyema, as in many other conditions, the surgeon's connection with the case is a late one, and the outcome is settled before he takes a hand. Within the month we have seen personally two such patients, who were treated medically for many weeks, and who were turned over to us at a time when the operative act was the final insult that could

not be endured. It can also be said fairly that neither physician nor surgeon could be expected to save some of these patients because the severity of the infection was such that their doom was sealed from the onset. Finally, and again with perfect fairness, we might say that certain patients in their series died because the surgeon, in taking over the case, took it over too completely; he overlooked the fact that the respiratory infection was by no means relieved, and he failed to retain the internist as his consultant—an error, it seems to us, that is all too frequent in too many diseases.

More than two-thirds of these cases were caused by the pneumococcus, either alone or in combination, and nearly 21 per cent by the streptococcus, which seldom, however, in civil practice reaches the heights of virulence which it did during the War. The location of the empyema seems to us of no special consequence, except in the single case in which the process was bilateral; fifty-six cases were on the right side and forty-three on the left side.

The duration of the illness naturally played a large part in the final outcome though it could not always be determined accurately, and the very long-standing cases were almost invariably tuberculous in character. It seems scarcely necessary to point out that a person who has been ill with a suppurative process for a long time, or who has been unwisely treated for it, enters the race with all the odds against him. In five cases the illness developed while the patient was in the hospital for other and totally unrelated conditions—in one case for a cataract operation under local analgesia—which leads us to remark again that it is never well to ignore the possibility of empyema, however impossible the circumstances may seem. In twenty-nine of the forty patients who died of toxemia and exhaustion, the illness prior to admission had lasted a month or more, usually without treatment. Among the children two had been ill for six and seven months, one with measles and whooping cough and the other with pneumonia, otitis media, and measles. In the same connection we might point out the high incidence of otitis media associated with empyema in children. Muller questions whether it plays any considerable part in the final outcome, but in six of the eight cases in children in whom it appeared in this series, we believe it was the one thing more which the exhausted little patients could not endure.

We are more convinced than ever, as the result of our study of these records, that repeated x-ray examinations should be a routine part of the treatment of empyema and that the x-ray findings should be utilized to the fullest in charting the plan of action. In some cases, particularly those treated by aspiration or by aspiration and air replacement, we do not think the x-ray was used enough; it was clear from the clinical course, particularly from the persistence of the pyrexia and from the respiratory embarrassment, that the suppurative process was being inadequately emptied, and x-ray evidence might have brought about a

change in the plan of treatment. In other cases, although the x-ray pictures clearly revealed inadequate drainage, no use seems to have been made of the finding. In a few cases, it must be admitted, the x-ray reports were misleading and suggested conditions within the chest entirely at variance with the clinical picture.

The backward look is never altogether fair. It is hard to be judicial in the absence of a full knowledge of all the facts, and it is frequently very unfair to pass judgment on the basis of what is written down, particularly when one is dealing with histories written only by internes, whose consciences, in this regard, are rather elastic. Some of these patients would certainly have died, no matter what was done for them. Some of them were undoubtedly tampered with too long, and for them the internist and the pediatrician are responsible. Some, if not all, of those upon whom emergency surgery was performed are the responsibility of the surgeon alone; he took a chance on their lives, and he, or rather they, lost the game. In many other cases the feeling is inevitable, even if not justified by the facts, that the result might have been different if something else had been done.

But we did emerge from this study with the conclusion that Hippocrates was right and that it would be well for us to return to the principles which he established many hundreds of years ago for the treatment of empyema, for they are fundamentally and permanently true. It is interesting that the study of the United States Army Empyema Commission confirmed the importance of his empirical observation regarding the treatment of this condition, and the best results are still achieved when the three rules he set down are carefully observed: that operation should not be performed early in the disease; that rapid evacuation of pus from the chest is almost inevitably fatal; and that the wound must be kept open until infection has subsided. Deaths would be fewer and chronicity markedly decreased if we forgot a great deal that has been written on the subject of empyema and returned to those three principles.

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HERNIA OF THE LUNG

Report of a Case, Outlining Mode of Fascia Lata Repair*

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ALTHOUGH the fear of respiratory failure and uncontrollable hemorrhage from manipulation of the lung is no longer a deterrent to chest surgery, operative procedure for lung hernia has received little standardization in the years following the Great War. It is true that experience is limited because hernia of the lung is rare; nevertheless, when it does occur, the conservative nonoperation is still advocated, despite the fact of its liability to recur after reduction by the closed procedure.[1, 2]

Goodman's survey[3] up to 1933, which includes an earlier study of the eighteenth century to 1925, comprises only 171 cases. Thus the reported incidence is low; yet, if complete statistical data are not available from our expeditionary forces in France, why should citations from the Russo-Japanese War, and even from the Civil War, be repeatedly brought up in our literature as evidence of the small proportion of hernias following trauma to the chest? Monographs on this subject are pregnant with erroneous statements made seemingly conclusive by many repetitions. Nomenclature is ambiguous because classifications are quoted from sources long prior to the birth of modern thoracic surgery. Moreover, we find a loose construction in terminology, for example, no differentiation between hernia and prolapse.

Hernia of the lung must not be confounded with prolapse, a classic example of prolapse being traumatic evisceration in the presence of a penetrating wound. Should a hiatus occur in the thoracic enclosure, as from a fracture of the rib, the lung may either prolapse or herniate, the latter form being designated in the event that the pneumonocele remains subcutaneous. Whether the parietal pleura remains unbroken or not has no bearing on terminology, although it naturally follows that in prolapse of the lung, as from a stab wound, the pleura would seldom fully encompass the protruding mass. But such a contingency must not imply a prolapse, since in our case to be reported a true lung hernia, the broken pleura, to quote Lilienthal,[4] did not permit "actual prolapse of the lung into the world through a wound." In the case of Montgomery and Lutz[5] the hernia, a consecutive type, occurred a year subsequent to recovery from a stab wound.

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Connors and Stenbuck[6] mirror the times by a statistical study, 1931-1932, of 68 penetrating stab and bullet wounds of the chest, though in no case was hernia of the lung an operative sequela; neither did prolapse follow the initial trauma. Under the category of "Thoracic Injuries," Hedblom[7] fails to designate lung hernia, though a series of 857 cases of diaphragmatic hernia is referred to. In an extensive monograph on surgery of the lung and pleura, Gask[8] makes no mention of hernia. We are in agreement with this authority that, aside from empyema, very few operations are done on the interior of the thoracic cavity. The surgeon confronted with the phenomenon of herniation must proceed on an entirely eclectic basis—periosteal plastic, rib resection, and other forms of closure to be discussed later. It is in the hope of contributing to the development of thoracic surgery that our operative procedure for hernia of the lung is presented, a case of which will be reported.

OPERATIVE TECHNICS

Technical details concerning actual entry into the thoracic cavity need not be discussed, since repair of the aperture is the problem pertinent to hernias. For closure of an orifice in the intercostal muscles Rose and Carless propose a single graft of fascia lata, covered by a pedunculated flap of muscle from the neighborhood.[9] Such transplantation is, however, less mobile than the weaving of fascia lata strips which, despite the fact that they do not stretch, permit complete mobilization of the chest wall for lung expansion. There should be no necessity of saving on repair tissue with a supply so easily and amply available.

Utilization of strips of fascia lata as sutures was introduced by Gallie and Le Mesurier in 1922, though prior to this McArthur had found that fascia from the external oblique retained its general characteristics when used in the repair of inguinal hernia.[10] In fact, it was the brilliant results achieved by the weaving of fascia lata in the inguinal type of persistent and recurring hernia that stimulated us to like application in the breach of lung hernia. Such a protective device lends itself well to the pathologic condition, and is valuable not only because applicable to large spaces but also because of its viability and inherent characteristics. It is evident that rib resection, a cumbersome method, fails to meet this requirement of full coverage. An osteochondral flap reflected on its periosteal hinge, as reported by Montgomery and Lutz,[5] is pronounced by Pain of Leeds University[1] as the most practical type of repair for hernias through the bony parts of the thoracic parietes. Nevertheless, in his own case the opening was considered too large for obliteration by this means, and consequently a defect measuring about three and one-half inches in its vertical diameter and two and one-half inches transversely was controlled by a corset with obturator. But control was not

so adequate as Pain affirms, since the patient was eventually compelled to discontinue work entirely.

Periosteal plastic closure has given good results in the hands of Goodman,[3] yet previous experience with the osteoplastic method has not always fulfilled the expectation of the surgeon, for nonunion may occur despite an apparently well-executed procedure. Although Graham[11] reports the successful issue of a case in which the hernial sac was closed by repeated gauze packings, this mode is not theoretically sound because of the liability of insufficient cicatrization and filling in, due to irritation from packing. Recurrence may well be prognosticated after palliative treatment, but should not be anticipated in the event of such radical procedure as hernioplasty.

From the foregoing critique it is evident that no one method has proved adequate as routine procedure for lung hernia. With woven fascia lata strips there should be no danger of failure to accomplish the desired result of permanent closure of the hernial opening. Successful surgery for lung hernia depends upon technical simplicity, adaptability to the pathologic condition, permanency, and viability of repair tissue. Since our technic with autoplastic sutures is in conformity with these principles of physiologic function, we believe that it will prove efficient in nearly all operable cases.

Technical Details.—Incision is made through the skin and subcutaneous fascia immediately over the tumor, or the equivalent of the pulmonary rupture manifested by tactile impressions. On exposure of the hernial orifice attention is first directed to removal of bone spicules, hemorrhage, and other intrapulmonary complications, prior to management of the pleural rent. Should the lung be fully covered by an unbroken pleura, Lilienthal's method is advocated for management of the pleura. At times a pleural defect may be of such extent as to preclude complete sutural coaptation, in which event closure is made as far as possible with plain catgut sutures, while the remaining portion of the rent is plugged by utilizing neighboring muscular structures (Fig. 3 *A* and *B*). For the purpose of lung expansion, since the lung is more or less in a state of collapse, positive pressure is exerted before tying the last stitch.

Repair proper is accomplished by covering the entire exposed area with strips of fascia lata introduced through both endothoracic fascia and periosteum, as demonstrated in Fig. 4. If a traumatized condition of the rib prevents inclusion of periosteum, good results can be expected by overtying the available endothoracic fascia alone. The actual detail of weaving these autonomous strips is identical with the method of approximating the internal oblique to Poupart's ligament in the management of indirect inguinal hernias. Should the major pectoral be ruptured, as illustrated in Fig. 2 *A*, overhand fascia lata sutures are desirable because of the added strength given this normal covering.

Chromic catgut is employed for suturing the subcutaneous fascial tissues, and clips are used for the skin. No drainage is provided.

CASE REPORT

W. H., a farmer about seventy years of age, was hospitalized March 16, 1934, suffering with painful respiration, obviously from a bulge on the chest wall. While at work on the farm he had fallen on a stump which struck him on the left side of the chest; as a consequence, the lump made its appearance before he had walked fifty feet.

A phase of his past history, while in no way signifying any pathologic condition of the ribs, is relevant to his present condition. When disembarking from a ferry in 1933 he had fractured the left second rib in the area of the recent injury; the ninth right rib had been fractured when logging thirty-five years previously.

Physical Examination. The patient, a poorly nourished and developed individual, suffered extreme pain on respiratory effort. Although the temperature was 98.6° F.,

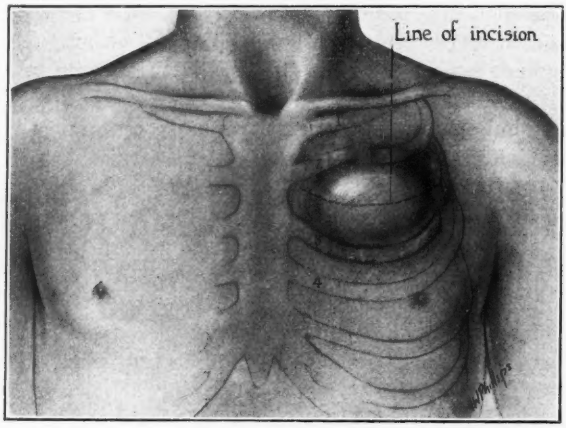

Fig. 1.—A pneumonocele, about four inches in diameter, evident during expiration, leaving a depressed area on inspiration, appearing and disappearing during speech. Note lines of fracture of second and third ribs, disclosed at operation, undemonstrable by roentgenograms.

pulse rate 78, respiration shallow, 16, and blood pressure 160/80 mm., his case fitted in with the conception of shock as understood in thoracic injuries, a fairly slow full pulse and an otherwise seemingly insignificant degree of shock. The anterior chest wall on the left side exhibited a marked depression in the region of the second and third intercostal spaces in the midclavicular line, a definite widening of the second interspace giving a feeling of patency. The chest wall bulged in and out during respiration, but abnormality of lung was especially evident during speech, when a rounded enlargement appeared about four inches in diameter, apparently of soft tissue (Fig. 1). At this date compression of the swelling evidenced no crepitation. On auscultation, breath sounds, though normal on the right, were covered with râles on the left, with râles scattered throughout the chest. Respiration was shallow and very painful on the left. Heart tones were normal with accentuated aortic second. No murmurs were heard.

Radiography, March 16. Although palpation of the depressed area of the chest indicated fracture of the second and third ribs, x-ray examination disclosed abso-

lutely no fracture signs. The stereo showed invasion of emphysema in the soft tissue of the left shoulder region but no evidence of a pathologic condition of the lung.

Subsequent emphysema, though not evident on earlier examination, was due to penetration of lung tissue by rib spicules, manifest by palpation of the inner aspect of the hernial opening.

Since the tumor appeared irreducible, it had been tentatively diagnosed as a strangulated hernia of the lung. After strapping with adhesive, gradual reduction of the mass was effected within a period of twenty-four hours.

Urinalysis, March 17. The urine was acid, specific gravity 1.020, and showed a very faint trace of albumin but no sugar—practically normal when considered with the patient's age.

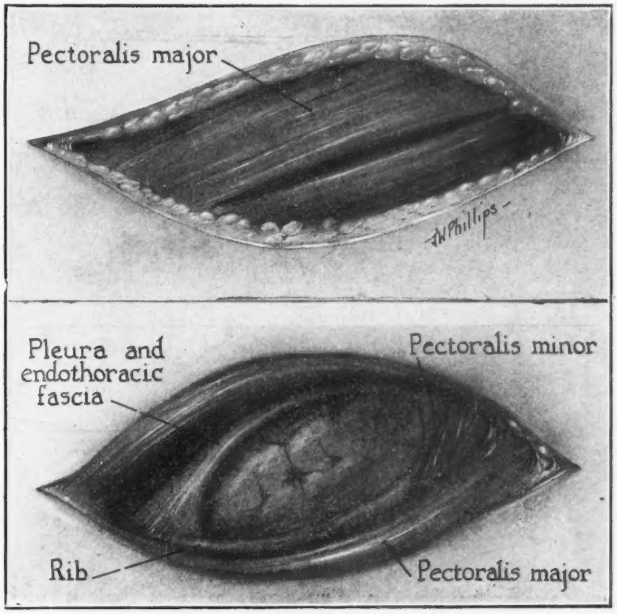

Fig. 2.—*A,* Laceration of the major pectoral, which permitted the considerable amount of herniation with partial strangulation.

B, Pleural defect disclosing the lung, delivered by positive pressure exerted by anesthetist. No adhesions were in evidence.

Final Clinical Diagnosis. Pleural rupture with fractured ribs.

Preoperative Course. The patient's condition was much improved by strapping, the pain becoming much easier. Further x-ray examination March 20 failed to confirm our clinical finding of fracture of the ribs. Nevertheless, some *locus minoris resistentiae* was present since the bulging in the chest wall recurred, a soft crepitant mass which proved refractory to local therapy. Because of recurrence of the hernia plus danger of necrosis from strangulation of tissue, surgical intervention was accordingly decided upon. Moreover, prognosis was becoming grave; pulse and respiration respectively 136 and 28, indicative of increasing thoracic shock. Expectant treatment in other hands has continually proved futile, and failure to rehabilitate is economically unsound.

Operation: On March 26, under nitrous oxide oxygen, the hernioplasty was done as previously described. Preliminary to surgery, morphine sulphate ¼ grain and atropine 1/150 grain were administered. Anesthesia was uneventful, untoward pneumothorax obviated by positive intrapulmonary pressure, maintained by intrapharyngeal insufflation.

Operative findings gave confirmation to clinical evidence: a 7 cm. rent in the upper fibers of the pectoralis major muscle immediately over the second interspace (Fig. 2 *A*); and fracture of the second rib near the chondral junction, and of the third at the axillary portion, with spicules of bone, at the fracture sites (Fig. 1). A 4½ cm. × 3 cm. pleural defect was disclosed between the second and third ribs, 9 cm. from the midsternal line (Fig. 2 *B*). On exposure of the hernial orifice the

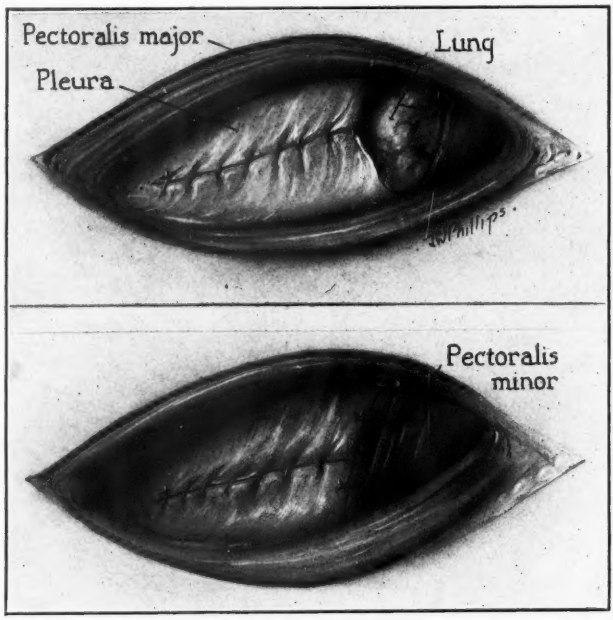

Fig. 3.—*A* and *B*, Closure of the parietal pleura. The torn edges were coaptated as far as possible; but the lateral half necessitated coverage by the pectoralis minor muscle, sutured in place as a plug, partial prevention against recurrence of the hernia.

lung was found free, with no adhesions of lung to pleura. There was no evidence of hemorrhage.

Repair of the pleural defect (Fig. 3 *A* and *B*) was as follows: Sutural approximation was continued as far as it was feasible; but over the remaining defect laterally, the immediately adjacent pectoralis minor was sutured to serve as a plug. Before tying the final stitch, differential pressure was exercised for lung expansion and consequent abolition of pneumothorax. Strips of fascia lata were then woven from the undersurface of the periosteum and endothoracic fascia of the second rib to the upper surface of the periosteum of the third rib, entirely covering the previous defect and muscle implant, and extending 2 cm. beyond on each side (Fig. 4 *A*). The rent in the pectoralis major muscle over the second interspace was repaired by overhand fascia lata sutures (Fig. 4 *B*). Closure of the subcutaneous

fascia was made with plain catgut, and clips to the skin. No drains were inserted. The patient was returned to bed in excellent condition, pulse of good quality.

Postoperative Course. During the first postoperative week the respiratory rate ranged from 22 to 24, falling to the 18-20 level during the following two weeks. The slight pneumothorax present, while accelerating the rate of respiration, maintained an air cushion entirely consistent with the healing process. Distress in breathing, and straining and coughing were allayed by administrating morphine sulphate, or codeine. Convalescence was comparatively easy.

Radiography of the chest, March 28, demonstrated a thickened pleura in the midaxillary line of the left lung field and an obliteration of the left costophrenic angle, with a small amount of fluid accumulation. The right lung appeared clear.

The patient was discharged April 12 after hospitalization of twenty-eight days, but returned for dressing April 19.

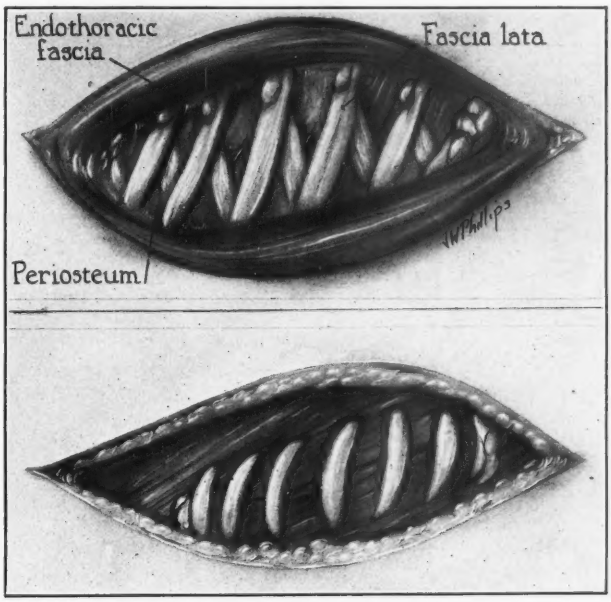

Fig. 4.—*A*, As a positive assurance of permanency, fascia lata strips were woven from the lower edge of the second rib to the upper edge of the third rib, each bite embracing both periosteum and endothoracic fascia. The entire site of injury to pleura, as well as that of the pectoralis minor plug, was covered by this weaving, which extended slightly beyond the limits of the hernial orifice.

B, Closure of pectoralis major rent by simple overhand fascia lata sutures.

June 8: Examination showed moderate chest expansion over the operated area, a definite firmness of scar tissue signifying obliteration of the intercostal defect. The patient was in excellent spirits, no sign of pneumonocele or distress symptoms. X-ray examination demonstrated complete lung expansion and normal structures throughout.

CONCLUSIONS

1. The operation as described for hernia of the lung is both safe and satisfactory when performed on properly selected cases, such conditions as tuberculosis and nephritis contraindicating surgery.

2. The validity of the operation is based on the following criteria: (a) stress is borne by the living suture material, not slowly stretching scar tissue of conjoined muscles; (b) weaving of fascia lata repair is quantitatively adequate.

3. Since the dread of entering the thoracic cavity has been removed, the economic aspect of lung hernia cases must be recognized; for debilitation of a patient, which may be expected under palliative measures, is inconsistent with modern thoracic surgery.

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EXPERIMENTAL OBSERVATIONS ON THE EFFECT OF 95 PER CENT OXYGEN ON THE ABSORPTION OF AIR FROM THE BODY TISSUES*

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THE absorption of air from the body tissues is governed by the law of gaseous exchange, which states that the rate of diffusion of a gas across a semipermeable membrane is proportional to the difference in the partial pressure of the gas on the two sides of the membrane. The actual time necessary for the absorption of a given volume of nitrogen from the body tissues depends on the surface area of the gas bubble, since only that fraction in contact with an absorbing surface can enter the surrounding tissues and from there the blood capillaries. For example, 500 c.c. of nitrogen in the intestine, the pleural cavity, or the peritoneal cavity will presumably require a considerably greater interval of time for absorption than an equal volume dispersed as tiny bubbles throughout the body, as in caisson disease.

The partial pressure of a bubble of nitrogen in the body tissues is 627 mm. of mercury, in comparison with a pressure of 573 mm. of mercury for the nitrogen in physical solution in the arterial blood.[1] This difference is responsible for the absorption of nitrogen from the tissues by the blood. The inhalation of pure oxygen instead of air results in a rapid reduction of the partial pressure of nitrogen in the blood stream. Thus, after one hour of breathing pure oxygen, the partial pressure of nitrogen in the arterial blood equals 155 mm. Hg; after two hours, 91 mm. Hg; after three hours, 52 mm. Hg; and after four hours, 31 mm. Hg.[1] Therefore, when pure oxygen is breathed, the diffusion pressure existing between a nitrogen bubble in the tissues and the nitrogen in the blood stream progressively increases, and one should expect a corresponding increase in the speed of absorption of nitrogen from the tissue spaces into the blood.

This is a report of an experimental study of the rate of absorption of air from the soft tissues of animals breathing room air as compared with others breathing pure oxygen.

METHOD

In each experiment a measured volume of room air was injected by a syringe and needle into the subcutaneous tissues of the thigh or into

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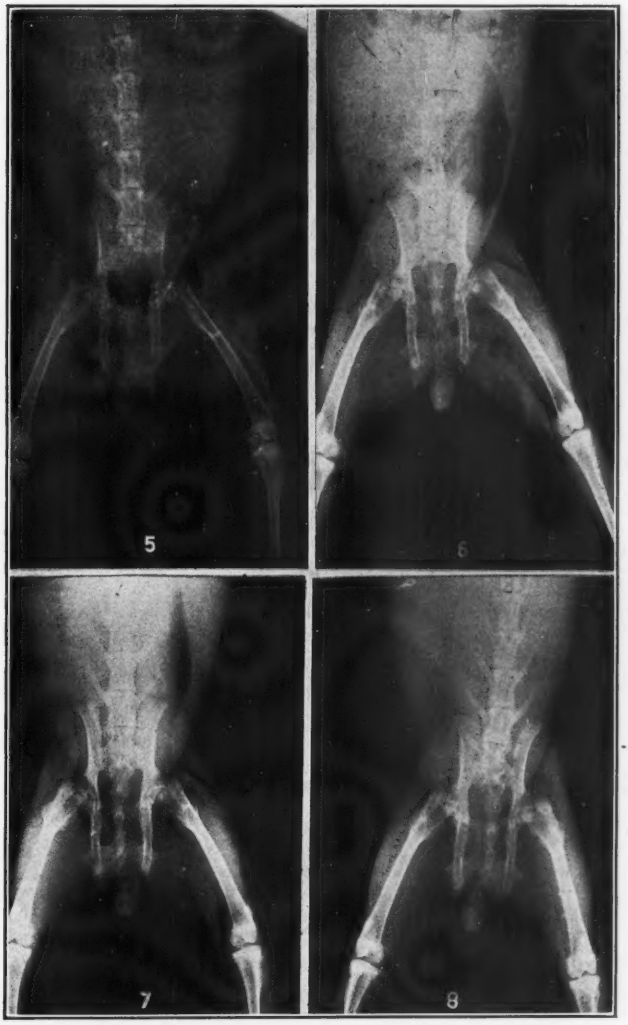
the peritoneal cavity of each of two rabbits. X-ray films were taken directly following the injection. One of the animals was immediately exposed to 95 per cent oxygen in an apparatus previously described,[2] while the other breathed room air. Exact quantitative determinations

Figs. 1, 2, 3, and 4.—X-ray films of rabbit taken at intervals after injection of 100 c.c. of air into the subcutaneous tissues of right thigh. *1*, Immediately after injection; *2*, after breathing room air for six hours; *3*, after breathing room air for twelve hours; and *4*, after breathing room air for twenty-four hours. Note the considerable volume of gas still present after twenty-four hours.

of the change in gas volume after any given interval would not only be difficult but unnecessary since for our purpose a sufficiently accurate approximation was obtained by serial x-ray films after six, twelve, and twenty-four hours.



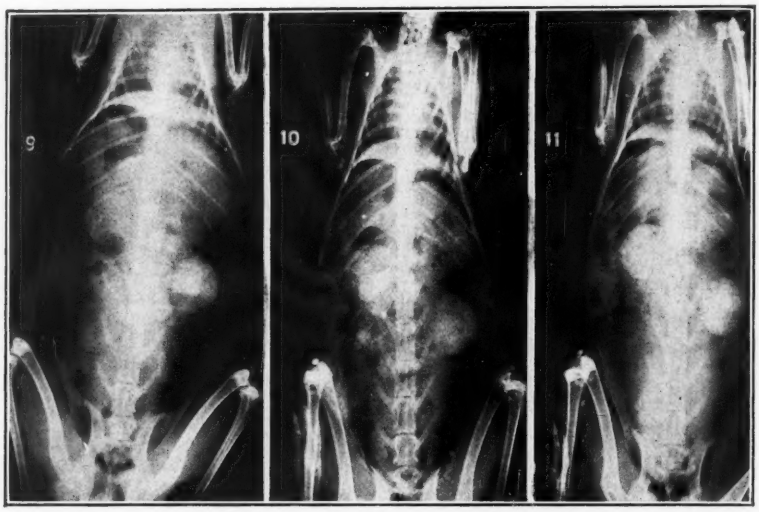
Figs. 5, 6, 7, and 8.—X-ray pictures of rabbit taken at intervals after injection of 100 c.c. of air into subcutaneous tissues of right thigh. *5*, immediately after injection; *6*, after breathing 95 per cent oxygen for six hours (note that already there is a decrease in the volume of gas in the tissues); *7*, after breathing 95 per cent oxygen for twelve hours (note a further decrease in gas volume in the tissues); and *8*, after breathing 95 per cent oxygen for twenty-four hours, when there is complete absorption of the gas. Note further by comparison with Fig. 5 the simultaneous disappearance of all the gas in the intestinal tract, an effect not observed in the animal breathing air (Figs. 1 to 4).

RESULTS

A. The Effect of the Inhalation of 95 Per Cent O_2 versus Air on the Rate of Absorption of Room Air From the Soft Tissues of the Rabbit's Thigh

Experiment IA.—One hundred cubic centimeters of air was injected into the subcutaneous tissues of the right thigh. The animal breathed room air. Figs. 1 to 4 show the x-ray films taken immediately after injection and after six, twelve, and twenty-four hours. The gas volume still present after twenty-four hours is considerable.

Experiment IB.—One hundred cubic centimeters of air was injected into the subcutaneous tissues of the right thigh of another rabbit. The



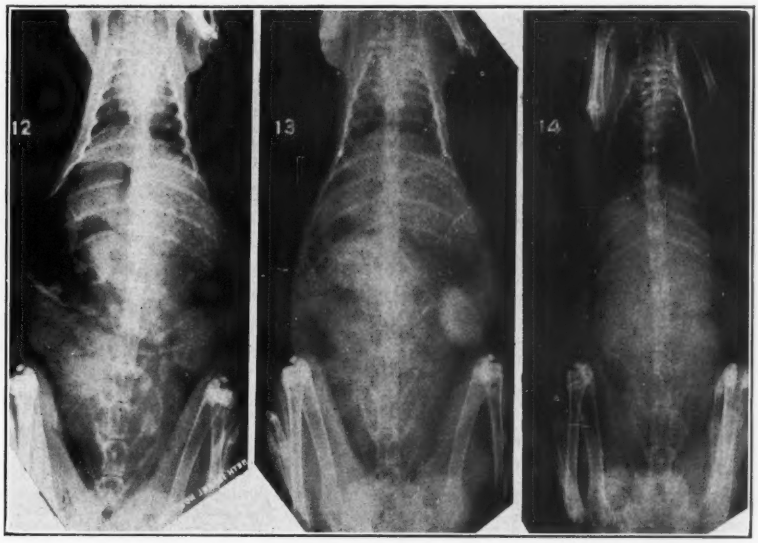
Figs. 9, 10, and 11.—X-ray films of rabbit taken at intervals after injection of 200 c.c. of air into the peritoneal cavity. *9*, Immediately after the injection; *10*, after breathing room air for six hours; and *11*, after breathing room air for twenty-four hours. Note that after twenty-four hours there is an appreciable volume of gas still left in the peritoneal cavity.

animal breathed 95 per cent O_2 for twenty-four hours except for short intervals during the taking of the x-ray films. Figs. 5 to 8 show the x-ray films immediately after injection and after six, twelve, and twenty-four hours. There was an appreciable absorption of air within six hours and a marked shrinking of the gas volume after twelve hours. The effect of oxygen after twenty-four hours is too obvious to require further comment. Incidentally, a comparison of these films with those of the rabbit breathing air (Experiment IA) demonstrates the simultaneous disappearance of all the gas in the intestinal tract, as well as in the thigh, of the animal breathing oxygen while in the one breathing air no marked change in the volume of intestinal gas can be made out.

These experiments were repeated five times with uniform results in all instances.

B. The Effect of the Inhalation of 95 Per Cent O_2 versus Air on the Rate of Absorption of Room Air From the Peritoneal Cavity of the Rabbit

Experiment IIA.—Two hundred cubic centimeters of air was injected into the peritoneal cavity of a rabbit breathing room air. Figs. 9 to 11 show the x-ray films taken immediately after injection and after six and twenty-four hours. There is a considerable decrease in the volume of air after six hours and still more after twenty-four hours, but a substantial amount still remains. There is a corresponding decrease in abdominal girth.



Figs. 12, 13, and 14.—X-ray films of a rabbit taken at intervals after injection of 200 c.c. of air into the peritoneal cavity. *12*, immediately after injection; *13*, after breathing 95 per cent oxygen for six hours (note decrease in size of abdomen and volume of gas in peritoneal cavity); *14*, after breathing 95 per cent oxygen for twenty-four hours. Note the almost complete absence of gas in the peritoneal cavity and intestinal lumen. Compare with Fig. 11.

Experiment IIB.—This is a repetition of Experiment IIA except that the animal breathed 95 per cent oxygen instead of air. Figs. 12 to 14 show the x-ray films taken immediately after injection and after six and twenty-four hours. That oxygen greatly accelerates the speed of absorption of the injected air is beyond dispute.

Four similar experiments using different volumes of air gave exactly the same results.

DISCUSSION

The simple principle involved in this effect of the inhalation of 95 per cent oxygen is applicable for the purpose of increasing the speed

of absorption of air (or nitrogen) which has been introduced or has gained entrance into any body cavity or tissue space. In conformity with this consideration we have in a previous publication[3] demonstrated that the gaseous distention of the obstructed small intestine, which is due primarily to nitrogen, can be reduced by some 60 to 70 per cent within twelve hours after the administration of 95 to 100 per cent oxygen, in contrast with a reduction of less than 10 per cent when the animal breathes air. This observation has been applied to patients suffering from distention of functional origin and has resulted in deflation of the intestine after a variable number of hours of administration of 95 per cent oxygen.[2] This effect we consider to be in direct consequence of the absorption of part of the distending gases with subsequent expulsion of the remainder by rectum as a result of a restoration of muscle tonus to the overdistended intestine incapable of effective peristaltic activity.

A further extension of the principle underlying the action of 95 per cent oxygen is the possibility of hastening the absorption of the air injected into the cranial cavity for encephalographic examination in order to avoid the distressing symptoms following this procedure. We are at present engaged in such a study and preliminary observations suggest that this will prove effective.

In the case of valvular pneumothorax or mediastinal emphysema, however, for which the method might be utilized, we encounter a different situation in that the volume of gas entering the pleural or mediastinal spaces is not a limited quantity, but continues to increase so long as the point of entrance of air remains open. Substituting pure oxygen for air as the respiratory gas would result in the absorption of the nitrogen, but its immediate replacement by the entrance of oxygen through the defect would offer no advantage in counteracting the pressure effects of the gas unless the rate of entrance of the oxygen through the defect were no greater than its rate of absorption by the tissue spaces.

The absorption of oxygen by the body tissues takes place rapidly, but under ordinary circumstances, with room air as the respiratory gas, nitrogen diffuses into the space occupied by the oxygen in the interval during which the oxygen is being absorbed. Fig. 15 shows the x-ray film of a rabbit breathing room air immediately after 200 c.c. of oxygen was injected into the right thigh. Fig. 16 shows the same rabbit four hours later. There is no significant decrease in the gas volume. On the other hand, when oxygen enters the body tissues and the animal is at once exposed to the inhalation of pure oxygen, nitrogen cannot diffuse in any substantial volume into the space occupied by the injected oxygen because it begins immediately to diffuse into the expired air. Thus Figs. 17 and 18 show an animal corresponding to that of Figs. 15 and 16, but breathing oxygen, at the beginning and at the end of four hours, respectively. The residual gas volume is considerably less.

Figs. 15 and 16.—X-ray pictures of rabbit taken at intervals after injection of 200 c.c. of pure oxygen into the subcutaneous tissues of right thigh, animal breathing air. *15*, immediately after injection; and *16*, after four hours. Note no significant decrease in the gas volume.

Figs. 17 and 18.—X-ray films of a rabbit after injection of 200 c.c. of pure oxygen into the subcutaneous tissues of right thigh, animal breathing 95 per cent oxygen. *17*, Immediately after injection, and *18*, after breathing 95 per cent oxygen for four hours. Note that the gas volume is considerably less in comparison with Fig. 17.

In consequence of these observations it may be inferred that the administration of oxygen for subcutaneous or mediastinal emphysema or for valvular pneumothorax should prove effective if the source of entrance of nitrogen (or air) has been sealed and may be of some help when the leakage of air is proceeding at a slow rate (i.e., slower than the rate of absorption of oxygen in an individual breathing pure or 95 per cent oxygen).

In a recent publication[2] we have described a simple and convenient apparatus for the prolonged administration of 95 per cent oxygen to patients. Our experience with this apparatus permits us to state that 95 per cent oxygen can, with a given technic, be administered for the purposes suggested above without encountering the toxic effects of this agent.

SUMMARY AND CONCLUSIONS

Theoretical considerations suggest that the absorption of nitrogen from the body tissues can be accelerated by the inhalation of 95 per cent oxygen. Experimental observations are reported demonstrating that this is the case for room air injected into the soft tissues and the peritoneal cavity of the rabbit. Previous studies have shown that this principle also applies to gaseous distention of the intestine in man and animals. We are at present engaged in a study of the effect of administering 95 per cent oxygen to patients submitted to encephalography for the purpose of relieving the symptoms following this procedure. Preliminary observations strongly indicate that oxygen will prove effective. Since the principle involved should be universally applicable for accelerating the absorption of air in any body tissue or space, we suggest that the inhalation of oxygen might be considered in the field of thoracic surgery whenever the problem of the disposal of encapsulated air arises.

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ARTIFICIAL PNEUMOTHORAX TREATMENT OF LOBAR PNEUMONIA*

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IN APRIL, 1921, Friedemann of Berlin reported a series of nine cases of lobar pneumonia which he had treated in 1919 by means of artificial pneumothorax. In this communication, the first of its kind, Friedemann stated that, although chronic pulmonary disease had been treated by means of artificial pneumothorax, acute disease of the respiratory tract had not previously been treated in this manner. Subsequently fifteen reports appeared in the literature, listing 86 cases of pneumonia treated by artificial pneumothorax. Of these 86 patients, 76 recovered and 10 died, a mortality rate of approximately 11.6 per cent. The earlier literature has been well summarized by Lieberman and Leopold.[1] Later reports by Behrend and Cowper[2] and Blake, Howard and Hull[3] are noteworthy.

The reported cases may be divided conveniently into five groups: First, there is Friedemann and the groups of workers stimulated by his efforts. Second, a group of pediatricians, notably Ibrahim and Duken, independently noted that in a pneumonic infant an accidental pneumothorax, produced while withdrawing fluid from an interlobar space, led to prompt amelioration of symptoms. These authors subsequently used the procedure when pneumonia existed without interlobar effusions and were favorably impressed by their results. The third group was stimulated by the work of J. J. Coghlan, who reported a series of 6 cases in the *Lancet* in 1932. Coghlan failed to mention the work of previous investigators. Fourth, Taylor in 1931 attempted to relieve the pain of lobar pneumonia by the introduction of oxygen into the pleural cavity. He treated three patients, in all of whom the pain was promptly relieved. Fifth, various American workers have adopted the procedure, applying it both to artificially produced lobar pneumonia in dogs and to the pneumonias of human beings. In general, the published reports have been strikingly favorable.

Through the interest of Harold Brunn and with the cooperation of LeRoy Briggs and Edwin Bruck of the Medical Service of the University of California Medical School at the San Francisco City and County Hospital and J. C. Geiger, director of the San Francisco Board of Health, certain patients with lobar pneumonia were treated by means

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TABLE I

	CASE	AGE	AREA	ENTRY	DISCH.	DAYS IN HOSP.	CULTURE		W.B.C.	DAYS TO CRISIS	DAYS TO LYSIS	DAY PNEUMO. BEGAN	TOTAL AIR	RE-FILLS	SYMP-TOMS RE-LIEVED	RESULT	COMPLICATIONS	VALUE
							SPU-TUM	BLOOD										
1	M. K.	46	L. L.	1/ 9	3/ 1	51	1	1	25,000		14	2	900	4	Yes	Recovery	Sterile effusion	Yes
2	D. B.	46	R. L.	2/ 8	2/12	5	1	1	12,500			1	500	2	No	Died	None	No
3	D. F.	23	R. L. + M.	1/25	2/ 8	17	15	0	12,000	8		2	500	1	Yes	Recovery	None	?
4	M. D.	59	L. L.	1/ 5	1/31	27	1	0	20,000		13	5	700	3	Yes	Recovery	NH_3 poisoning	?
5	P. H.	53	R. L.	1/30	2/24	26	1	0	17,000		11	2	950	4	?	Recovery	None	Yes
6	H. Y.	22	L. L.	1/22	3/ 1	39	1	0	14,000		16	1	850	2	Yes	Recovery	Sterile effusion	Yes
7	H. T.	42	R. L. + M.	2/12	2/28	17	15	0	11,300	6		2	700	2	Yes	Recovery	None	Yes
8	R. M.	31	R. L. + M.	1/20	1/22	3	1	0	25,500			2	150	1	No	Died	Pregnant	No
9	W. R.	50	L. L.	1/15	2/10	27	1	0	16,300		17	2	300	1	Yes	Recovery	None	Yes
10	E. S.	33	R. L. + M.	2/10	3/29	47	1	0	21,400			3	750	3	Yes	Recovery	Empyema	No
11	W. M.	36	L. L.	2/14	3/28	42	1	0	18,000			2	300	1	No	Recovery	Pyopneumo-thorax	No
12	J. G.	40	R. L.	3/13	3/30	17	2	0	18,700		11	2	1,475	4	Yes	Recovery	Sterile effusion	No
13	T. L.	39	R. U. + M.	3/13			6	6	23,500			1	1,200	3	Yes	Died	Meningitis	No
14	F. T.	44	R. L.	4/ 9	4/26	18	1	0	20,600	7		2	1,725	5	Yes	Recovery	Syphilis	Yes
15	F. O.	25	R. L.	4/20	4/28	8	16	0	23,280	2		1	700	2	Yes	Recovery	None	Yes
16	J. F.	45	R. L. + M.	5/ 2	5/ 5	3	1	1	15,500			1	525	2	No	Died	None	No
17	J. D.	29	L. U.	8/ 7	8/20	13	?	0	11,360		3	1	950	3	Yes	Recovery	None	?
18	J. B.	44	R. U.	8/24	9/18	25	1	1	9,680	1		6	350	1	Yes	Recovery	None	?
19	H. H.	49	R. L.	1/27	–	–	14	0	19,750	10		8	2,050	5	Yes	Recovery	None	Yes
20	P. N.	21	L. L.	1/16	–	–	1	0	15,650		8	8	1,200	4	Some-what	Recovery	Spread to sound lung	?
21	L. S.	34	R. L.	10/22	11/13	23	1	1	12,240		7	4	700	2	No	Recovery	Hydropneumo-thorax	?
22	A. M.	24	L. L.	12/17	–	–	IV	0	18,800	8		4	1,500	4	Yes	Recovery	None	Yes

NOTE.—Cases 3, 8, and 15 were females. Nos. 19, 21 and 22 still in hospital (Feb. 5, 1935). Relief of symptoms refers to pain and dyspnea.

of artificial pneumothorax at the San Francisco City and County Hospital beginning in the winter of 1933-34. Up to the present time, a total of 22 cases of lobar pneumonia have been treated by this means. Three of the patients were females; 19 were males. They ranged in age from twenty-two to fifty-nine years. In the majority of instances the pneumonia was on the right side, and in 6 patients more than one lobe was involved. The sputum revealed Type I pneumococcus in 14 cases, Type II in 1 case, Type VI in 1 case, Type VII in 1 case, Type XV in 2 cases, Type XVI in 1 case, Type XIV in 1 case, and Type IV in one case. In 5 patients with Type I pneumococcus in the sptum there were positive blood cultures; 2 of these patients died. In all there were six positive blood cultures with three deaths. Four of the 22 patients treated by pneumothorax died, a mortality of 18 per cent, which is a little less than one-half of the total mortality during the period under discussion. It should be noted that 2 of the patients died within a few hours after pneumothorax was induced. They were dying at the time the procedure was undertaken. A complete summary of cases treated is given in Table I, with a clinical impression of the value of treatment in each case.

Those who have had the opportunity to observe these patients have reached few conclusions regarding the value of this form of treatment in pneumonia. The impression has been gained that pain can be promptly relieved by compression of the affected lung, and that the disadvantages of pain relieving drugs, strapping and counterirritants can be avoided. We believe that the toxemia may be diminished in certain instances and the subsequent course changed for the better. It is our impression, however, that once air is introduced into the pleural cavity, it should be given for at least two weeks to allow the inflammatory reaction in the pleura to subside. Otherwise, sterile effusions, which prolong the patient's stay in the hospital, may be expected to develop. Aside from one case of empyema, the result, we believe, of technical difficulties easily avoidable, we do not think that any patient was harmed by the induction of pneumothorax.

We wish to express our appreciation to Dr. Wm. J. Kerr, Professor of Medicine, University of California Medical School, for his helpful suggestions and criticism, and to Dr. Charles Sirocco, Resident in Medicine, University of California Medical School at the San Francisco City and County Hospital, for his unfailing interest and assistance.

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THE APPLICATION OF THE ZIPPER RUBBER DAM IN LOBECTOMY*

H. Brodie Stephens, M.D.
San Francisco, Calif.

INFECTION of the pleural cavity is one of the main causes of mortality and morbidity following the operation of one-stage lobectomy. To protect the pleural cavity from gross contamination we have em-

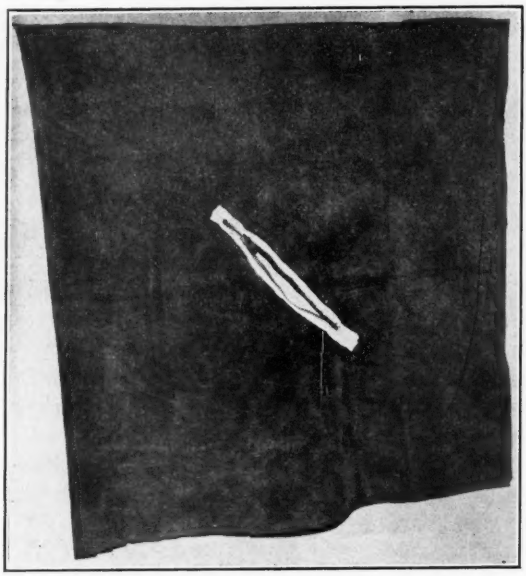

Fig. 1.—The rubber sheet pictured above measures 24 inches square. The zipper opening measures 7½ inches and is simply stitched onto the rubber.

ployed for several months a large sheet of No. 26 rubber dam. A zipper is sewed into the center of the square rubber sheet. (Fig. 1.)

When the lobe to be removed has been freed up to its pedicle, the zipper is opened wide and the rubber sheet is then threaded over the lobe. The zipper is now pulled up so that the opening is just sufficient to permit the pedicle to pass through. (Fig. 2.) The rubber sheet is then

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draped over the operative incision and thus completely extrapleuralizes the division of the pedicle. Following the closure of the pedicle stump the

In Fig. 2 the rubber sheet has purposely been shown not to cover the entire operative wound in order better to picture its application. Two Roberts-Nelson tourniquets surround the pedicle, and the latter is severed between them.

rubber sheet is removed; the operator and assistants change their gloves before proceeding with the completion of the operation.

Department of Reviews and Abstracts

Conducted by Duff S. Allen, M.D., Associate Editor

Selected Abstracts

Ochsner, Alton; Gage, Mims; and DeBakey, Michael: Scalenus Anticus (Naffziger) Syndrome, Am. J. Surg. **28**: 669, 1935.

Scalenus anticus syndrome is a clinical entity, the symptoms of which are identical with cervical rib symptoms. The symptoms are the result of compression of the brachial plexus and subclavian artery on the first thoracic rib. The compression may be due to an abnormally low position of the shoulder, to high fixation of the sternum and ribs, to low origin of the brachial plexus, and to elevation of the first thoracic rib due to spasm of the scaleni muscles resulting from brachial plexus irritation. The first three conditions are predisposing; the last is an exciting factor. Irritation and stimulation of the brachial plexus by pressure of the first rib, causing spasm and shortening of the scalenus anticus muscle, result in elevation of the first rib, thus establishing a vicious cycle.

Microscopic section of the scalenus muscle removed at operation revealed degeneration and replacement of muscle fibers by connective tissue.

Pain is the most constant symptom of this syndrome. It is often referred to the shoulder and down the arm. Tenderness over the scalenus anticus muscle is fairly constant.

This syndrome occurs oftener in women than in men. The authors report six cases, all of them women. In four cases the symptoms were on the right side, in two cases on the left.

Vascular changes are frequently present but are usually missed unless oscillometric studies are made. These vascular changes consist of diminution of pulse volume on the affected side, decrease in surface temperature, numbness, coldness, etc. Sympathetic stimulation is undoubtedly responsible for many of the vasomotor changes.

Scalenus anticus syndrome must be differentiated from cervical rib syndrome, subacromial bursitis, supraspinatus tendon rupture, cervicodorsal sympathalgia, Raynaud's disease, and brachial plexus neuritis.

The only treatment which promises a cure is resection of a portion of the scalenus anticus muscle.

Henry L. Cabitt.

Davies, H. Morriston: The Treatment of Cavities in Pulmonary Tuberculosis, Brit. J. Tubere. **29**: 91, 1935.

The author discusses the pathology and effects of cavity formation in tuberculosis. The more centrally a cavity is placed in the lung, the more favorable it is for its healing. A cavity in the apex is in an adverse position. Peripheral cavities often extend to the pleura and cause adhesions, with resultant futile attempts if pneumothorax therapy is tried. Thus the nearer a cavity is to the surface of the lung, particularly in the apex or the interlobar septum, the earlier must active treatment in the form of artificial pneumothorax be begun.

Small cavities may close with bed rest. An exudative cavity over 3 cm. in diameter or a productive cavity over 2 cm. in diameter should have artificial pneumothorax therapy.

The author discusses the beneficial combination of artificial pneumothorax and phrenic operations. He advises phrenic operation for cases in which pneumothorax has been tried but has failed to produce collapse.

The prospect of collapse by phrenic operation in larger cavities is so doubtful that it should be done only as a preliminary to thoracoplasty or apicolysis. He discusses the various types of thoracoplasty, apical, complete, and anterolateral.

Henry L. Cabitt.

Clerf, Louis H.: Subcutaneous Emphysema as a Complication of Foreign Body in a Bronchus, Ann. Otol., Rhin. & Laryng. **44**: 364, 1935.

The author reviews the literature on the spontaneous occurrence of subcutaneous emphysema in a case of foreign body in a bronchus, prior to bronchoscopy or other surgical procedures.

He reports two cases of bronchial obstruction due to foreign body and complicated by subcutaneous and mediastinal emphysema. Both patients had extreme emphysema involving the head, neck, and chest; in both the obstruction was due to a peanut, and in both the symptoms cleared after removal.

The mechanism of the formation of this subcutaneous emphysema is not known since there have been no autopsy reports. The theory expounded is that, since obstructive emphysema is common in vegetal foreign body cases, in the presence of severe paroxysms of coughing, the distended air vesicles may rupture forcing air into the lung tissues. This air is forced along the hilum of the lung into the medi-

astinum, thence through the upper aperture of the thoracic cage to the tissues of the face, neck, and trunk. The extent and degree of the subcutaneous emphysema is in proportion to the effectiveness of the valvular action of the foreign body.

Upon removal of the foreign body, the air contained in the bronchi cannot be forced through the ruptured alveoli; the subcutaneous emphysema subsides, and the air in the tissues is promptly absorbed.

Henry L. Cabitt.

Wright, R. Douglas: Reaction of Pulmonary Tissue to Lipiodol, Am. J. Path. **11**: 497, 1935.

The author reviews the literature on the reaction of tissues to lipoid substances. He reports a case of lipiodol in the lung of a patient twelve months after instillation. This case came to autopsy and microscopic sections of the lung revealed the lipiodol enclosed in a foam of cellular syncytium. These cells, the lipophages, have their origin probably from the supporting tissue of the bronchi. No epithelial reaction occurs.

There was also noted a thick hyaline tissue on the diaphragmatic pleural surface, which was a peculiar form of granulation tissue. Scattered throughout this area were macrophages, some containing small droplets of lipiodol. This tissue is unique and is probably a reaction to lipiodol.

Henry L. Cabitt.

The American Association for Thoracic Surgery

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The American Association for Thoracic Surgery

The eighteenth annual convention of the American Association for Thoracic Surgery was held in New York City on June 3, 4, and 5.

This year for the first time there was official representation from the Society of Thoracic Surgeons of Great Britain and Ireland. Mr. J. E. H. Roberts of London spoke on "Diagnosis and Treatment of Chronic Empyemata." Mr. H. Morriston Davies of Ruthin, North Wales, discussed "General Principles of the Surgical Treatment of Pulmonary Tuberculosis."

The formal program given by members of the Association again was very inclusive. The surgical treatment of pulmonary tuberculosis, the treatment of carcinoma of the esophagus, lobectomy for bronchiectasis, pulmonary embolectomy, and various related subjects were dealt with extensively. All of these papers will appear in subsequent issues of the JOURNAL OF THORACIC SURGERY.

An unusually instructive series of clinics was given at the various hospitals in New York City.

The following officers were elected:

President	Dr. Carl Eggers	New York, N. Y.
Vice President	Dr. Leo Eloesser	San Francisco, Calif.
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Dr. John Alexander	Ann Arbor, Mich.
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The following were elected to honorary membership:

Dr. William Lerche	Cable, Wis.
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Mr. H. Morriston Davies	Ruthin, North Wales
Dr. Rudolf Matas	New Orleans, La.
Dr. Howard Lilienthal	New York, N. Y.

The following associate members were elected to active membership:

Dr. William E. Adams	Chicago, Ill.
Dr. J. B. Amberson	New York, N. Y.
Dr. Norman Bethune	Montreal, Canada
Dr. B. Noland Carter	Cincinnati, Ohio
Dr. Cameron Haight	Ann Arbor, Mich.
Dr. Thomas J. Kinsella	Oak Terrace, Minn.
Dr. Richard Overholt	Boston, Mass.
Dr. Leo Rigler	Minneapolis, Minn.
Dr. Carl Steinke	Akron, Ohio

The following was elected directly to active membership:

Dr. William F. Rienhoff	Baltimore, Md.

The following were elected directly to associate membership:

Dr. Clarence E. Bird	Louisville, Ky.
Dr. Howard H. Bradshaw	Boston, Mass.
Dr. Samuel Friedlander	Cleveland, Ohio
Dr. Howard Gray	Rochester, Minn.
Dr. Edward M. Hanrahan	Baltimore, Md.
Dr. Jerome Head	Chicago, Ill.
Dr. Wm. Francis Honan	New York, N. Y.
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Dr. W. L. Rogers	San Francisco, Calif.
Dr. Sidney Shipman	San Francisco, Calif.
Dr. John Strieder	Ann Arbor, Mich.
Dr. Grant Thorburn	New York, N. Y.
Dr. Arthur S. W. Touroff	New York, N. Y.
Dr. Wm. M. Tuttle	Detroit, Mich.

The nineteenth annual meeting of the American Association for Thoracic Surgery will be held in Rochester, Minn. The dates for the meeting will be determined later.

Medical

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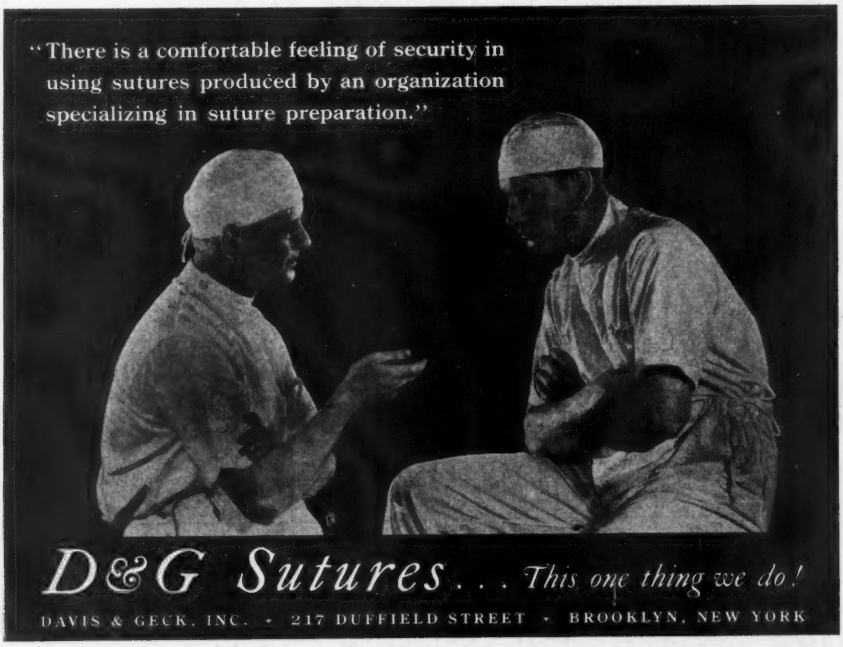
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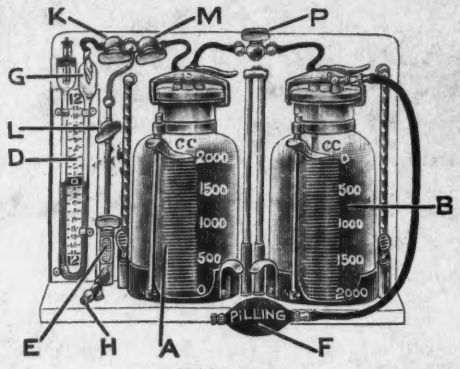

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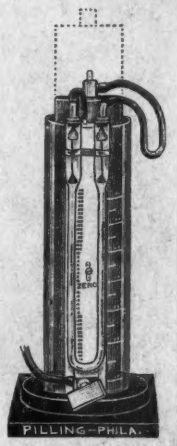

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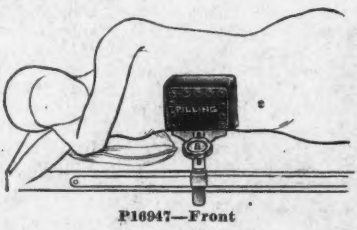

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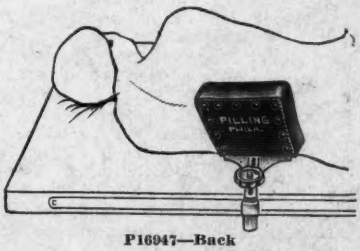

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